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THE ALLERGIC CHILD*

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Of necessity this paper must deal with the general rather than with the particular; so the term 'child' must include any young human, male or female, from birth to the age of 16 years. This upper age is included to allow reference to be made to a few of the complications arising under the influence of adolescence. Perhaps the foetus should be included too, for if we could find some prophylactic treatment for our potentially allergic baby the work and worries of parents, nurses and health visitors would be eased considerably. Various authors have given figures varying from 10 to 75% for inheritance of allergy, but any survey must depend upon the thoroughness of our delving into family medical histories and upon our ever-widening knowledge of the range of allergic manifestations in the different organs of the body.

A recent outstanding contribution to the study of inheritance in allergy is that by Schwartz (1952). He concluded that asthma is an inherited disease and that a genetic relationship exists between asthma and vasomotor rhinitis, Besnier's prurigo and hay-fever. To Schwartz it seems possible but not very probable that urticaria and angioneurotic oedema in females may in certain instances be genetically related to asthma, but that eczema, migraine, psoriasis, gastro-intestinal allergy, ichthyosis and epilepsy are not. Schwartz considers the age of onset of asthma to be independent of inherited predisposition and rejects the theory of Wiener, Zieve and Fries (1936). Ratner and Sullivan (1953) reviewed the work of Schwartz and of others and from this and their own material concluded that no genetic hypothesis fits all the data. In the words of D. A. Williams of Cardiff, 'The present position appears to be much confused and worthy of deep thought'. Bowen (1953) over a period of 15 years observed 59 pairs of identical twins. He found co-existing allergies in twins to be the exception. In 52 cases the allergic condition existed in only one twin and in only 7 instances was there a true bilateral allergy of similar pattern.

An Experiment in Prevention

Twelve years ago 40 pregnant mothers who were all known sufferers from allergic asthma were persuaded to undergo a 6 months' course of desensitization during their pregnancy with the hope of desensitizing the foetus too and so avoiding the production of more allergic children. Most of these mothers were so pleased with their healthy babies that they insisted, in subsequent pregnancies, on further detailed investigation and desensitization. Up to date the 40 mothers have produced 93 children, with only 3 cases of obvious allergy amongst them; many of these children are already 11 years of age. Even the most sceptical statistician would have expected that 15 would have developed eczema or hay-fever by this date. However, this really proves nothing; we must treat it as a long-term experiment and review the same families 20 years hence.

The First Year of Life

Let us proceed to the infant in its 1st year of life. It is known that in early infancy the Schick test is unreliable as a guide to the level of immunity to diphtheria; but Payling-Wright and Clark (1946) have shown that the low reactivity of new-born infants' skins to diphtheria toxin is not due to any insensitivity to histamine or to inability to exhibit the typical triple response on injury. They state, 'Since similar anergy is found in other forms of delayed response at this age, such as that produced by ultra-violet light, the mechanism underlying these responses may differ from that concerned with immediate reactions, and may not become fully functional until some weeks after birth. It seems likely that the lesser skin reactivity seen in delayed reactions in infants is partly due to the relatively rich and superficial vasculature of their skins, which would facilitate the escape both of injected substances and of vasodilator products of injured tissues'. So much for

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

the delayed immunological reactions; but it is just this rich vasculature which makes it necessary, when looking for the *immediate* response to allergy tests in a young infant, to use the 'prick test' and to take one's readings after 3 minutes, not, as with adults, after the usual 10 minutes.

From Tables I and II (cases 1 and 2) it will be seen that, given the right technique and the right solutions, it is possible to get the correct answer to tests on babies of 3-6 weeks, and that these food allergies can be confirmed at 6 months and at 1 year of age. Both these

TABLE I. CASE 1. A.L. ECZEMA OF FOREHEAD AND CHEEKS AT 6 WEEKS. PRICK TESTS.

	6 *Weeks*	6 *Months*	1 *Year*
Milk	—	—	—
Egg	++	+++	+++
Orange	—	—	—
Fish	—	—	—
Wheat	—	—	?+
Meats	—	—	—

TABLE II. CASE 2. BABY S. GENERALIZED ECZEMA FROM 2 WEEKS. ASTHMA FOLLOWING A HEAD COLD AT 3 MONTHS (JUNE). PRICK TESTS

	3 *Weeks*	3 *Months*	1 *Year*
Milk	—	—	—
Egg	—	—	—
Fish	—	—	—
Orange	—	—	—
Mixed vegetables	++	+	++
Parsnips	+++	+++	+++
Other vegetables	—	—	—
Pollens	—	++	++

babies were breast-fed and one must presume that the egg protein and the parsnip protein were ingested by the mother and that the allergenic part of the molecule was transmitted in the milk. The mother of baby S. (case 2) gave a history of being excessively fond of parsnips and of eating them daily.

Under what circumstances are we going to attempt to find an allergy as the cause for ill-health in our infant and young child? Manifestations of allergy may be *obvious*, as in eczema or asthma, or may be *obscure*, as in cyclic vomiting, in early migraine, or in gastro-intestinal allergy, sometimes called indigestion, but all too often labelled acidosis for want of a better word.

OBVIOUS ALLERGIES

Let us consider the *obvious* allergies first. Many doctors when confronted with an irritable, cross-tempered child, rubbing and scratching its own skin, a misery to itself and its parents, or with a hunch-backed, pigeon-chested, wheezing child, now think in terms of allergy or of psychology, but seldom realize the combination of the two. Shakespeare must have had some experience of these children, to write with such feeling in his 'Seven Ages of Man': 'At first, the infant, mewling and puking in the nurse's arms. Then the whining school-boy, with his satchel and shining morning face, creeping like snail unwillingly to school'.

Which comes first, the skin irritation or the bad-temper? There can be any possible combination from 100% allergy and 0% psychology to 100% psychology and 0% allergy. From cases 3-6 you will see that after removal of the offending allergen, the personality changes rapidly for the better; perhaps the allergy was more than skin-deep, and directly responsible, originally, for the temperament; but so far we have no proof. However, we must decide that all such children should be investigated and if possible told what to avoid.

Case 3. W.P., male, aged 2½ years. Normal babyhood except slight eczema, which developed when aged 6 months and gradually became more widespread during the next 2 years. Considered by mother to be a bad-tempered baby compared with his 2 elder brothers. Some scarring of face and neck due to scratching. Allergy tests: *chocolate* +++, *beans* ++. Five treatments with 2 c.c. histamine by electrophoresis 1 milliamp for 10 minutes, at the rate of 2 doses per week, needed to control irritation (Walker 1954). Advised to continue avoiding chocolate and beans in diet and come for desensitization by injection when aged 6. If eczema due to some other food develops during the next few years, this patient should have another course of histamine treatment immediately.

Case 4. A.W., female, aged 3. Mother described her as a very healthy, contented baby, normal in every way, until weaning was begun at 6 months. Gradually became difficult to manage, with indigestion, poor appetite and sleeplessness. By 8 months was covered with an itching eczema. At 2½ years was a puny child with hard dry skin and some haemorrhagic patches due to scratching. Owing to fact that trouble began at weaning, the mother had tried goat's milk with some success but had still used cow's milk in cooking puddings. At this stage allergy tests showed: *Cow's milk* ++++, *cheese* ++++, *fish* +++. Six treatments with histamine by electrophoresis at the rate of 3 a week for 2 weeks stopped the irritation, healed the skin, and apparently reduced the degree of allergy, for a re-test showed: *Cow's milk* ++, *cheese* ++, *fish* −.

Case 5. E.S., female, aged 6 years. Infantile eczema since 4 months old, tending to be worse in winter. Very difficult, ill-tempered child, especially since going to school 1 year ago. Other children said she must have 'fleas' because she scratched her face, neck and back until blood appeared on her fingers. Allergy tests: *Chocolate* +++, *fish* +++. After avoiding those foods for 4 weeks all lesions had healed, but now she was teased because she could not eat her school dinners. Ten treatments with histamine during the Christmas vacation (3 weeks) have reduced her allergic state to a minimum. She now eats fish and chocolate, but has been advised to avoid the routine cod-liver-oil next winter.

Case 6. D.E., male, aged 3. *Slight eczema* since 6 months old, now getting much worse and localized to eyelids and back of neck. Irritation, especially at night. Only time ever clear was when upstairs in bed with high temperature, on fruit juices only. Allergy tests (17 February 1953): *Cow's milk* ++, cat fur ++, dog hair +++. Given goat's milk in diet and animals removed from home. On 17 March 1953 symptom-free. Skin clear for first time since 6 months old. Sleeps through the night. On 8 August 1955, still symptom-free.

Occasionally one is confronted with a child less than 3 years old with very definite spasmodic asthma, but on the whole it is the eczematous baby that is the potential asthmatic of the future. Can we, by treating the allergic state in the infant prevent, wholly or in part, the development of asthma at a later date? By the age of 6 many children are already missing weeks or even months of schooling each year owing to asthma. In a survey carried out during the war years on a number of children from the Oxford elementary schools, tested and treated for their asthma between the ages of 6 and 10 years, it was found possible to get an almost asthma-free population in the secondary schools (aged 12-15), and in my opinion it is the duty of every parent, every family doctor and specialist, to cooperate in every way to enable this handicapped child to overcome his allergic state before he reaches the more competitive age of schooling; for, as soon as he realizes that owing to

some inborn disability he cannot compete with his colleagues in sport and enjoy all the fun of life to the full, a gradual neurosis overtakes him. If for some unfortunate reason he has not been treated at a younger age, he will need prolonged help from his medical advisor or a specialist in psychology before he faces the world squarely again.

A boy in his teens is usually brought to see me by one or both of his parents, and the first step towards giving him confidence in handling his own abnormality, which should be treated as a temporary disability rather than an illness, is to see him alone, let him give his own history, and hear how his troubles affect him. Then he is tested with a full range of inhalants with which he may have contact, pollens of plants growing in his district or country, and foods which he eats regularly. These all give the immediate or extracellular type of reaction within 10 minutes and, if positive, are playing some part in spasmodic attacks of asthma. If, however, these tests are all negative and from the history one suspects the gradual onset of long bouts of asthma, then tests must be done with common bacterial proteins and common household and atmospheric moulds. Occasionally these give an immediate response, but the site of the tests should be examined after 24 hours, for bacterial allergy is often of the *delayed intracellular* rather than the *immediate extracellular* type. Children from 12 to 16 should themselves be made responsible for avoiding any foods or animals to which they have given a positive test, and encouraged in every way to co-operate with their medical advisers. Most parents are only too pleased to agree to this, but a few will continue to 'fuss' over every suspected wheeze.

OBSCURE ALLERGIES

In contrast to the obvious allergic manifestations, there are many conditions in which allergy is only one of a number of causes to be considered in the differential diagnosis. Foremost among these is a syndrome of poor appetite, marked underweight for age, and lack of 'pep' sometimes, but by no means always, associated with a spotty skin. These children may be and often are allergic to some common food, which can be avoided in the diet, and no other treatment is required; occasionally the same syndrome presents as a seasonal trouble, usually in the spring or summer and is really just one more possible way of showing pollen sensitivity. A common fallacy with doctors as well as parents is to expect pollen sensitivity to show always as the classical hay-fever with rhinitis and conjunctivitis, but this is far from the truth. During the summer of 1955 in Oxford, cases at the Clinic showed pollen allergy manifesting as:

1. Rhinitis and conjunctivitis (2-16 years)
2. Conjunctivitis only, with no nasal symptoms whatever (4-16 years)
3. Asthma with rhinitis
4. Asthma with conjunctivitis.
5. Asthma, without any nasal or ocular signs
6. Weeping eczema (aged 3) from 8 June until controlled with antihistamines on 10 July; after 20 July no antihistamines needed.
7. Vomiting and diarrhoea in a child of 5 from 8 June to 20 July
8. Mucous colitis in a boy of 15 from 8 June until 31 July
9. Scleritis and episcleritis without rhinitis (aged 6 years)
10. Iritis without conjunctivitis (aged 8 years)
11. Headaches with occasional *petit mal* between 4 June and 20 July 1955 (aged 5 years). Mother reported same symptoms in June and July last year).

Other examples of obscure allergies are shown in the cases 8-11:

Case 8. J.C., male (7 years). Frequent left supra-orbital headaches with visual disturbances and vertigo during last 2 years. Grandmother had migraine, mother has rheumatoid arthritis. Allergy tests (14 December 1954): *Beef* ++, *mutton* ++, *pork* +. Avoided meats for 2 months and remained symptom-free. 12 February 1955, desensitization by injection begun (6 weeks). 9 August 1955, has been eating meat since March, still symptom-free.

Case 9. P.B., male (9 years). Chronic 'sticky' conjunctivitis each summer since 3 years old. Periodic deafness, worse in summer since otitis media when 4 years old. Skin irritation each June and July since age of 7. Conjunctival swab—*Staph. aureus* sensitive to penicillin. After treatment with penicillin in March 1954 all symptoms disappeared until 1 June 1955, when irritation of skin appeared. 4 June 1955, conjunctivitis. 6 June 1955, deafness. Allergy tests: *Grasses* ++, *house dust* ++, *fish* ++.

Case 10. S.R., female (10 years). 'Gritty' eyes and some stickiness for 6 months. 11 July 1955, conjunctival swab negative. Allergy tests: *Horsehair* +++, *feathers* +++. Had been sleeping on feathers and had a pet bird. These avoided for 1 month. 9 August 1955, reported symptom-free since 14 July 1955.

Case 11. R.C. (4 years). Folliculosis of conjunctiva June 1954 and June 1955. No infantile eczema; no true hay-fever of eyes and nose. Mother had migraine, uncle had corneal ulcers. Allergy tests, prick method (21 June 1955): *Cocksfoot grass* +++, *rye grass* ++, *Timothy grass* ++. Antistin-privine eye-drops and Benadryl elixir given: clear in 2 weeks.

Now that rheumatologists and allergists are agreed that allergy plays some part in rheumatic fever and a large part in the aetiology and treatment of gout and of rheumatoid arthritis, we must realize that swollen joints of children's diseases may be, but are not necessarily, due to an allergic response to an endogenous virus, or may be imitating the hydrarthrosis of adults caused by exogenous factors such as inhalants and foods. This year Lewis (1955) has published evidence supporting a close relationship between Schonlein-Henoch purpura, acute haemorrhagic glomerulo-nephritis and acute rheumatism, and considers hypersensitivity to be a major aetiological factor in all three conditions.

MANAGEMENT OF THE ALLERGIC CHILD

This must be considered under two headings: (1) during an allergic attack, and (2) during other illnesses.

During an allergic attack, whether of the short sharp type, as in spasmodic asthma, or of the prolonged type, as in chronic eczema, every effort must be made to shorten the attack and so prevent permanent tissue-damage. The use of antihistamines during the last 10 years has helped those suffering from eczema, hay-fever, oedema of sinuses or of joints, to control the acute stage until investigation for the offending allergens can be completed, though it is generally agreed even by the manufacturers that antihistamines do not relieve the asthmatic. It is possible and desirable to give a small dose of adrenaline by injection to babies from 3 months of age to control true asthma but 6 minims of 1/2000 adrenaline for a baby of under 1 year is probably more than enough. If adrenaline be given *slowly*, at the rate of 1 minim per minute, there is no danger of shock;

but 1/4 tablet of neo-epinine under the tongue is often easier for the doctor than a 6-minute injection.

In all allergic conditions the primary aim must be to find the cause of the attack. Skin tests have a useful place in our scheme, though they are not infallible. It is difficult to assess the result unless one knows from much practice the capabilities of one's solutions and does adequate controls. Experience is really the best guide. Far too many doctors have ordered a few vials of testing solutions, of doubtful age and origin, from some commercial firm and found that every test gave a positive reaction or every one gave a negative, and so condemned skin tests as useless. One gains confidence in one's technique only when the results obtained can be repeatedly confirmed by clinical trial. Even then, such results must be assessed together with the history and the development of the allergic condition and with any other positive findings on thorough examination of the patient. For comparison one should remember that although only one doctor in 100 is capable of taking and reading an X-ray plate, the other 99 do not condemn radiology.

It is all-important to realize that every child in an allergic state may have a general reaction to any new drug; so it becomes necessary to carry out preliminary intradermal tests before a full dose is given, care being taken not to obscure the result by an earlier dose of antihistamine. Antibiotics should be avoided wherever possible since they often act as sensitizers, turning any minor allergy present into a major one and so producing unexpected complications in a child who is already ill. In very severe illness, when some antibiotic is really necessary to save life, yet a preliminary test shows an allergy to the drug, it is wise to give some antihistamine by injection immediately before each dose of the antibiotic, but the careful balancing of the inter-reactions of the two injections must be considered by the doctor in charge every few hours, for it is quite impossible to write up any pre-arranged dosage charts since the requirements of any two patients are never exactly similar.

Some children, though very few, should have tonsils and adenoids removed, but our allergic child is very seldom among this list. Wet, boggy, pale nasal mucous membranes, oedematous adenoidal tissue, enlarged but non-septic tonsils, are never successfully treated by surgery. The offending allergen is usually an inhalant dust or pollen, but not always, for a common food (milk, wheat, fish, etc.) was the main cause of chronic non-seasonal nasal catarrh in 23 out of a recent series of 60 school-children between the ages of 6 and 12 years. In each case the results of skin-testing were confirmed by avoidance of the suspected foods for 3 months before any treatment by desensitizing injections was ordered. Allergy, if under control, need never be a contra-indication to tonsillectomy where this is obviously necessary to avoid frequent bouts of tonsillitis or mechanical interference with speech or breathing. Similarly, our allergic child need not be denied vaccination against small-pox or immunization against diphtheria or yellow-fever, provided these are undertaken only after tests with horse serum, egg white or other substrate used in the preparation of the immunizing fluids.

In conclusion, one would like to predict that in the near future not only every paediatrician but every doctor, will have been introduced to the study of allergy in the clinical pathology department of his medical school, for surely allergy is nothing more nor less than abnormal physiology; and that he will appreciate the interdependence of allergy and psychology in his training in children's wards and out-patient departments.

SUMMARY

After a brief summary of the recent literature on the inheritance of an allergic diathesis, a preliminary report of an experiment to prevent the development of inherited allergic manifestations in the newborn is presented. This is followed by an account of results of 15 years' work with children from 0 to 16 years. Allergies are divided into groups: (1) *obvious*, such as eczema, asthma, urticaria or conjunctivitis, and (2) *obscure*, such as chronic indigestion, cyclic vomiting, migraine, mucous colitis, acidosis, convulsions, or *petit mal*; and it is shown that no child is too young to be investigated and, if found allergic to some common food or inhalant, told how to avoid it. From the age of 7 years onwards every child is encouraged to understand the stresses and strains of everyday life, especially as they influence his own disability. He himself as well as his parents must realize how the psychosomatic nature of his attacks increases with his years and will change, for better or for worse, with the approach of adolescence. It should be the aim of every paediatrician, who must be in part a psychologist and in part an allergist, to overcome during the childhood of his patients any allergic condition of whatever degree, and so be able to consent to the vaccination of an eczematous baby, to immunization against diphtheria and other killing infections, or to the removal of infected tonsils where necessary; and also to reassure the parents that the later development of alternating allergies is no longer a potential shadow over his approaching adult life.

The management of an allergic child in health and disease is discussed with special reports on the influence of sensitizers, such as the antibiotics.

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EDITORIAL

HEPATIC ENCEPHALOPATHY

'Appolonius at Abdara suffered for a long time without taking to his bed. He had an enlarged abdomen and a pain in the region of the liver to which he had become accustomed, for he became jaundiced, flatulent and of pallid complexion.

'As a result of eating beef and drinking cows' milk, he developed what was a slight fever at first and went to bed. He got much worse through taking a large amount of milk—and a generally bad diet. He began talking at random, showed loss of memory in anything he said, and became disorientated.

'About the fourteenth day—his temperature rose and he went out of his mind; there was shouting, disturbance and much talking, then he settled down again and relapsed into coma. Subsequently his bowels were upset. The excreta were not always the same; sometimes they were small in quantity and dark and rust-coloured or they were greasy, raw and pungent.

Thirty-fourth day: died.'

Hippocrates: Epidemics, Book III (xiii).[1]

Cerebral complications of liver disease have been recognized by physicians since antiquity. Modern literature includes references to the writings of Galen,[2] Boerhaave[3] and Morgagni[4] on this subject, and we quote above a case history from the works of Hippocrates which has particular interest in the light of recent concepts of the pathogenesis of this disorder.

The encephalopathy under consideration is a distinct entity and has been referred to as 'hepatic coma'[5] or as 'porto-systemic encephalopathy'.[6] It is to be distinguished from a number of other syndromes of cerebral disorder to which patients with liver disease are prone, e.g. Korsakoff's psychosis, Wernicke's encephalopathy, pellagrous dementia, subdural haematoma, intolerance of barbiturates, Kinnear Wilson's disease, and so on. It occurs most frequently in cases of hepatic cirrhosis and in severe infective or toxic hepatitis. It has also been reported with chronic obstructive jaundice, eclampsia, and abscess or metastatic carcinoma of the liver. In cirrhotic patients the syndrome is most often precipitated by haemorrhage from the upper alimentary tract. Diuretics, particularly ammonium chloride and ammonium-containing exchange-resins, abdominal paracentesis, intercurrent infection and acute alcoholism may also act as precipitants.

In fulminating hepatitis, the clinical picture usually develops rapidly; in the cirrhotics, however, a more

VAN DIE REDAKSIE

HEPATO-ENCEPHALOPATHIA

,Vir 'n lang tyd het Appolonius in Abdara gely voordat hy gaan lê het. Sy buik was vergroot en hy het 'n pyn in die lewerstreek gehad waaraan hy gewoond geraak het want hy het geelsug, winderigheid en 'n bleek gelaatskleur ontwikkel.

,As gevolg van die beesvleis wat hy ge-eet het en die koeimelk wat hy gedrink het, het hy 'n ligte koors gekry en is hy bedtoe. Te wyte aan van die groot hoeveelheid melk wat hy gedrink het—en 'n swak dieet oor die algemeen—het sy toestand baie vererger. Hy het deurmekaar begin praat, geheueverlies getoon en het verward geword.

,Ongeveer die veertiende dag—sy koors het gestyg en hy het van sy verstand geraak; hy het geskreeu, was oproerig en het baie gepraat, toe het hy weer bedaar en in 'n koma verval. Sy maag het begin werk . Die ontlasting was nie altyd dieselfde nie; somtyds was dit min en donker en roeskleurig, of dit was vetterig, rou en skerp.

Vier en Dertigste dag: gesterwe.'

Hippocrates: Epidemics, Book III (xiii).[1]

Serebraalkomplikasies wat op lewersiekte volg is al eeuelank aan geneeshere bekend. Hedendaagse literatuur oor hierdie onderwerp bevat verwysings na Galen[2] Boerhaave[3] en Morgagni[4] en hierbo het ons 'n gevallegeskiedenis uit die werke van Hippocrates aangehaal, wat besonder interessant is met die oog op die jongste opvattings in verband met die ontstaan van hierdie siekte.

Hierdie harsingaandoening is 'n duidelike entiteit en is na verwys as *hepatic coma*[5] of as *portosystemic encephalopathy*.[6] Dit moet onderskei word van 'n aantal ander sindrome van harsingstoornisse waartoe pasiënte met lewerkwale geneig is, bv. Korsakoff se psigosie, Wernick se harsingaandoening, *pellagrous dementia*, subdurale hematoom, onverdraagsaamheid i.v.m. barbiturate, Kinnear Wilson se skiekte en dies meer. Dit kom mees dikwels voor by gevalle van lewersirrose en by ernstige besmetlike of toksiese lewerontsteking. Dit is ook al aangeteken gepaard met kroniese verstoppende geelsug, stuipe en verswering of metastatiese karsinoom van die lewer. In sirrotiese pasiënte word die sindroom veral verhaas deur bloedstorting uit die boonste spysverteringskanaal. Uriendrywende middels, veral ammonium chloried en uitruilingsharssoorte wat ammonium bevat, buikparasentese, tussenkomende infeksie en akute dranksug kan ook die toestand verhaas.

As die lewerontsteking heftig is, ontwikkel die kliniese beeld gewoonlik snel; met sirrose is die proses gewoonlik meer gestadig. Kennisse van die pasiënt sal verandering in sy persoonlikheid bespeur; die vrolike, dranksugtige sirrotiese pasiënt word nors en kwaai; sy persoonlike gewoontes word slordig; hy ly aan nagmerries en sy

insidious onset is common. Those acquainted with the patient will observe a change in his personality; the cheerful, bibulous cirrhotic becomes morose and truculent; his personal habits become slovenly; he suffers from nightmares and his demeanour may vary from extreme lethargy to acute mania. In the established case, the hiccupping, grimacing or yawning patient characteristically lies in bed with his legs crossed and his knees drawn up onto his abdomen. He is often rowdy and may be violently restless. Consciousness may be simply clouded, sometimes with reversed sleep-rhythm, or the patient may be stuperose or in deep coma. Muscle twitchings or choreiform movements may be present, but most typical is the 'flapping tremor' first described by Adams and Foley[5] and likened by Sherlock[6] to the beating of a bird's wings. To elicit this sign the patient's hands are held outstretched in front of him and rapid irregular, asymmetrical flexion-extension movements of the wrists and metacarpophalangeal joints are observed. Further examination reveals variable, asymmetric spasticity of either pyramidal or extrapyramidal type, occasionally with extensor plantar responses. Roving eyeballs, dilated pupils, grasping or sucking reflexes and hyperpyrexia are other inconstant signs.

When liver function tests are made in these cases, biochemical disturbance is almost always demonstrated. The changes may be relatively slight and do not correlate in any linear fashion with the degree of neurological disorder. A more constant finding of great interest is the disturbance of ammonia metabolism,[7] manifested by an abnormally high ammonia-concentration in the peripheral blood and the cerebrospinal fluid. This finding provided the clue to the pathogenesis of the disorder.

It has long been known that dogs with 'Eck fistulae' (i.e. experimentally produced porto-caval anastomoses) develop neurological abnormalities when fed on meat.[8] In human beings with severe liver disease or with porto-caval shunts, similar disturbances may follow the ingestion of ammonia-containing substances.[9] Sherlock and her associates[6] have shown that these substances pass unmetabolized through the damaged liver into the systemic circulation or may actually by-pass the liver *via* porto-caval collaterals. The resulting excess of ammonia interferes with the delicate processes of cerebral metabolism and neurological symptoms ensue. Riddel[10] has suggested that ammonia disturbs the Kreb's citric-acid cycle by combining with α-ketoglutaric acid to produce glutamic acid; the latter is further aminated to produce glutamine, of which a considerable excess has been found in the cerebrospinal fluid of patients with hepatic encephalopathy.[11]

The primary pathogenic factor, therefore, is faulty metabolism of the nitrogenous material absorbed from the bowel, and a resemblance to the mellow concept of 'auto-intoxication' will be apparent. This is particularly striking in the recently recognized syndrome of 'chronic intermittent hepatic coma',[3] in which exacerbations of a chronic course are related to occult gedrag mag van uiterste letargie tot akute manie wissel. In gestaafde gevalle is dit kenmerkend om die hikkende, grynsende of gapende pasiënt in die bed te kry met sy bene gekruis en sy knië tot op sy maag getrek. Hy is dikwels lawaaierig en mag geweldig onrustig word. Hy mag net beneweld wees, somtyds met sy slaappatroon omgekeer, of die pasiënt mag in bewusteloosheid of in 'n diep koma versink. Spier- of senuweetrekkings mag aanwesig wees maar die mees tiperend is die ‚flapbewing' wat vir die eerste 6 keer deur Adam en Foley[5] beskryf is en deur Sherlock vergelyk is met die flap van 'n voël se vlerke. Om hierdie teken aan die lig te bring word die pasiënt se hande voor hom uitgestrek en vinnige, onegalige, asimmetriese buig-strekbewegings van die polsgewrigte en die middelhand-vingerlitte word waargeneem. Nadere ondersoek stel aan die lig wispelturige, asimmetriese spastisiteit wat òf primidiaal òf buiteprimidiaal is, af en toe met voetsoolstrekspierreaksies. Ronddolende oogballe, oopgerekte oogappels, klouende of suigende reflekse en hoë koors is ander tekens wat somtyds waargeneem word en ander tye nie.

Wanneer die lewerwerking in hierdie gevalle getoets word, word biochemiese stoornisse byna altoos ontdek. Die stoornisse mag betreklik gering wees en korreleer geensins lynvormig met die graad van neurologiese ongesteldheid nie. 'n Meer bestendige bevinding van groot belang is die steuring in die ammoniummetabolisme[7] wat geopenbaar word deur 'n abnormaal hoë konsentrasie ammonium in die perifeerbloed en in die harsing en rugmurgvloeistof. Hierdie bevinding is die leidraad tot die patogenese van hierdie ongesteldheid.

Dit is al vir 'n geruime tyd bekend dat honde met 'Eck fistulae' (d.w.s. poortholte-anastomose wat eksperimenteel verkry word) neurologiese abnormaliteite ontwikkel as hul op vleis gevoer word[8]. By pasiënte met ernstige lewerkwale of met poortholte-anastomose kan soortgelyke steuring op die inname van ammoniumbevattende stowwe volg.[9] Sherlock en haar medewerkers[6] het gevind dat hierdie stowwe sonder stofverwisseling deur die beskadigde lewer na die grootbloedsomloop passeer of dat dit selfs die lewer vermy en die ompad via die poortholte-kollaterale kies. Die oormaat ammonium wat hierop volg belemmer die delikate prosesse van serebraalmetabolisme en neurologiese simptome volg daarop. Riddel[10] meen dat die Kreb-sitroensuurkringloop verstoor word deurdat die ammonium met α-ketoglutaarsuur verbind om glutamiensuur te vorm; laasgenoemde word verder ge-amineer om glutamien te vorm waarvan 'n oormaat in die harsing en rugmurgvloeistof van pasiënte met hepato-encephalopathia gevind word.

Die belangrikste patogeniese faktor is derhalwe die gebrekkige metabolisme van die stikstofhoudende materiaal wat van die ingewande geabsorbeer word, en hierin word 'n ooreenkoms met die ou opvatting van selfvergiftiging ge-openbaar. Dit is besonder opvallend in die sindroom van ‚kroniese onderbroke hepatiese koma'[3] waarin 'n opvlamming van die kroniese verloop in verband staan met verborge of duidelike maagdermbloedstorting, maaltye van 'n hoë proteïengehalte, of inname van ammoniumsoutpurgasies.

Hierdie nuwe kennis van die ontstaan van die siekte beïnvloed nou die behandeling van die akute toestand.[12]

or obvious gastro-intestinal haemorrhage, to high-protein meals or to the ingestion of ammonium salts.

Treatment of the acute condition has been modified by this new understanding of its pathogenesis.[12] Restriction of nitrogenous intake and the inhibition of endogenous protein metabolism are regarded as fundamental. To this end, all nitrogen-containing substances—protein, amino acids and ammonia-containing medicaments—are eliminated from the diet. Bleeding oesophageal varices are occluded as quickly as possible by balloon tamponage,[13] and bacterial activity in the gut is retarded by large oral doses of a tetracycline antibiotic. Further 'de-amination' of the gut is attained by repeated enemata and purges of magnesium sulphate. Endogenous protein metabolism is depressed by a high oral or parenteral carbohydrate-intake and by vigorously combating any infection. The patient's fluid and electrolyte requirements are sedulously maintained; supplemental vitamins, including vitamin K, are administered and sedation, when necessary, is achieved with paraldehyde. Intravenous transfusions of whole blood for post-haemorrhagic shock or anaemia are not contra-indicated. Finally, when clinical improvement occurs, protein is gradually brought back into the diet and the patient's tolerance is assessed. Most cases of hepatic cirrhosis can take about 50 g. of protein daily without neurological upset.

Walshe has postulated that glutamic acid converts the noxious ammonia into inert glutamine, and that a relative deficiency of glutamic acid occurs in ammonia intoxication. He therefore treated a small group of cases of hepatic coma with glutamic acid and reported good results.[14] Several other workers have failed to confirm these results,[10] but Ridell[9] is of the opinion that glutamic-acid therapy may benefit some cases of hepatic cirrhosis with encephalopathy.

The prognosis of this condition until recently was uniformly dismal. Encouraging results with the therapeutic regime outlined above, however, have emphasized the need for the more frequent and earlier diagnosis of the disorder.

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Die beperking van die inname van stikstofhoudende stowwe en die onderdrukking van proteïenmetabolisme wat inwendig ontstaan, word nou as fundamenteel beskou. Met hierdie doel voor die oë word alle stikstofbevattende stowwe—proteïen, aminosure en ammoniumbevattende geneesmiddels—uit die dieet geskakel. Bloedstortende slukdermspatare word so spoedig moontlik deur middel van ballontampons afgesluit[13] en die werking van bakterië in die derm word vertraag deur groot mondelinge dosisse van 'n tetrasiklien-antibiotika. Die derm word verder ‚ont-amineer' deur herhaaldelike enamata en purgasies van magnesium sulfaat. Endogene proteïenmetabolisme word onderdruk deur 'n hoë mondelinge of buitedermse koolhidraat-inname en deur kragdadig die hoof aan enige infeksie te bied. Die pasiënt se vloeistof- en elektrolietbehoeftes word sorgvuldig in voorsien; aanvullende vitamiene, veral vitamien-K, word toegedien en indien kalmering nodig is word paraldehied gebruik. Daar is geen teenaanwysing nie vir binneaarse bloedoortapping met heel bloed om bloedarmoede of skok wat op bloedstorting volg te bestry nie. Ten slotte, wanneer kliniese verbetering intree, word proteïen geleidelik in die dieet teruggebring en die pasiënt se dulding daartoe getoets. Die meeste gevalle van hepatiese sirrose kan omtrent 50 g. proteïen daeliks inneem sonder enige neurologiese stoornisse.

Walshe postuleer dat die glutamiensuur die skadelike ammonium in logge glutamien omskep en dat 'n relatiewe tekort aan glutamiensuur by ammoniumvergiftiging voorkom. Hy het derhalwe 'n klein groep pasiënte met hepatiese koma met glutamiensuur behandel en goeie resultate noteer.[14] 'n Heel paar ander navorsers kon hierdie resultate nie staaf nie[10] maar Ridell[9] meen dat glutamiensuurterapie sommige pasiënte wat ly aan hepatiese sirrose met encephalopathia kan baat.

Tot onlangs was die prognose vir hierdie kondisie deurgaans somber. Die belowende resultate wat verkry word met die behandeling wat hierbo uiteengesit is, beklemtoon die noodsaaklikheid om die ongesteldheid meer dikwels en vroeer te diagnoseer.

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IRITIS OF ALLERGIC ORIGIN*

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In England the study of allergy in ophthalmology has been developing for the last 25 years, but it was not until 1947 that the results were sufficiently well established to justify a general discussion at the Royal Society of Medicine. At that meeting Mr. Gayer Morgan[1] (senior ophthalmic surgeon at Guy's Hospital) reminded us that almost every disease of the eye had been reported as allergic in origin in some particular patient. Since then the literature on the subject has been copious, but vague, and can be more easily understood if the word 'disease' is deleted and replaced by 'condition'.

To me[2] an allergic condition is acute in onset and, if recognized and treated at once, will clear up quickly, often within a few minutes or hours, leaving no permanent damage to the tissues involved; but it must be recognized that once a tissue has remained in an abnormal physiological condition for a longer time, as in recurrent keratitis or iridocyclitis, there may be secondary changes, due perhaps to pressure of oedema, perhaps to inflammation, perhaps to secondary infection, which must be healed by routine treatment and may leave permanent scarring.

ALLERGY IN GENERAL

The term 'allergy' is not in the English dictionary. It was first used by a Frenchman to describe an 'altered reaction', not just hypersensitivity to some drug or food but a changed type of reaction; thus, if one gets a dizzy headache after 1/10 gr. of quinine instead of after 25 gr. then one is *hypersensitive* to quinine, but if after the 1/10 gr. one gets not a headache but a spasm in the chest, an attack of gout, or a painful iritis, then one is *allergic* to quinine.

Many workers have considered an allergic reaction as a pathological state similar to the abnormal antibody-antigen relationship of bacterial immunology, but this cannot explain the simplest allergic response or immediate reaction as seen in hay-fever, urticaria, or a bee-sting; in these states a slight biochemical variation, possibly congential, in the serum alters its ability to permeate the walls of the capillaries allowing them to 'leak' and so produce the watery secretion of hay-fever or the fluid of oedema in enclosed spaces.

These reactions are *extracellular*, in contrast to the delayed or *intracellular* reactions of contact dermatitis, of asthma, or of rosacea keratitis. In extracellular allergy sensitivity can be transferred passively by plasma; in the delayed or intracellular type whole cells or their contents are required.

By using the fluid from an allergic oedematous swelling of one patient for an intradermal test on another known allergic patient, it is possible to satisfy oneself that free-histamine has been assembled in the fluid in this one organ; that is to say, the metabolism or distribution of histamine throughout the body has been upset and it has been collected in the 'shock tissue' of the moment, be it skin, lung, bladder or iris. From an attempt to show that histamine was still the offending substance in the more delayed type of allergic reaction a method of using a dose of free histamine for the diagnosis of the intracellular allergies due usually to some chronic bacterial or virus infection in a distant organ has been developed and was published in the *Acta allergologica* for 1954.[3]

To explain why one organ, be it eye, ear, chest or stomach, is selected as the 'shock tissue' to carry the full responsibility of an allergic attack takes us into the realm of metaphysics; but as it need not be the eye as a whole, but rather the conjunctiva, cornea, lens or iris alone, which may be concerned in any one patient, the simplest belief is that some localizing previous injury, either developmental or traumatic, is necessary. This idea would certainly help to explain a unilateral allergic condition in one of two symmetrical organs, and one-sided headaches after a motor-cycle crash.

ALLERGIC IRITIS

What part do these ideas play in helping in the investigation and treatment of iritis? If one remembers that the iris is a 'diaphragm of blood vessels and unstriped muscle fibres held together by a very loose spongy stroma' (Parsons and Duke-Elder), one cannot fail to recognize an almost ideal setting for an acute anaphylactoid reaction. That this reaction can be of either the immediate extracellular type or of the delayed intracellular type is illustrated in the 6 case-histories summarized below. They also serve to support the suggestion of Professor Pickering[4] that in the 'immediate response' group, histamine or some histamine-like substance is released and the effect can be neutralized by anti-histamines but not by cortisone, as happens in hay-fever or in angio-neurotic oedema; while in the delayed group, as in bacterial allergy in other tissues, there is an intracellular reaction which can be overcome by cortisone but not by antihistamine. *Cortisone does not cure: it only suppresses the mechanism of reaction.*

One practical point to remember is that when anti-histamines are used in the treatment of iritis they must be supplied to the body by mouth or by injection, for no amount of antistin-privine slopped on to the conjunctiva will reach the iris in sufficient concentration to do any good. All patients with recurrent attacks of acute iritis should have a full range of allergy tests as part of the routine hospital investigations, for 20% of all iritis is due to allergy, the offending allergens being foods, inhalants, drugs, or toxins (including tuberculin).

Case 1. G.L., male (60). 1942-1950—14 attacks iritis. 1950—All investigations negative except allergy tests: horsehair +++, dog hair ++. Avoidance and antisin given and attack cleared in 8 days; desensitized by injection. 1955—Reported no iritis since 1950.

* A paper presented at the South African Medical Congress, Pretoria, October 1955.

Case 2. V.B., female (46). Recurrent iritis with urticaria. 1949—Meibomian swab, chest and sinus X-ray, teeth, urine, Mantoux test, blood count, ESR and WR all N.A.D.; allergy tests: house dust ++, grass pollen +++; desensitized by injection and remained symptom-free until: 1954—Iritis but no urticaria; re-test: house dust ++, grass pollen, ++; desensitized again. September 1955—Still symptom-free.

Case 3. B.S., female (33). 1950—First attack iritis, cleared up after 8 weeks with hot bathing and rest. 1951—Iritis cleared up in 5 weeks with cortisone, bathing and rest. 1952—Referred to Allergy Clinic: beef +++; healed in 1 week with antistin and avoiding beef. 1953—Ate beef in error: acute iritis, healed in 3 days with antistin tablets. 1955—Symptom-free for 2 years; still avoiding beef.

Case 4. R.N., male (51). Recurrent iritis since 1947. 1941—Tuberculous glands removed from neck. 1947—First attack iritis; all usual investigations negative. Frequent attacks iritis until 1951—All tests repeated: Had become atropine-sensitive. 1955—Healed by cortisone; allergy tests all negative except O.T.1/100,000 +++; desensitized by 10 injections O.T. Retest: O.T. 1/10,000 +; all quiet so far.

Case 5. G.B.L., male (57). 1940-1950—16 attacks iritis. 1951—Iritis and spasmodic bronchitis; all investigations, including allergy tests, negative except *Strept. viridans* protein ++++; treated with cortisone; healed in 3 weeks; auto-vaccine from sputum given for 6 months. 1955—Still symptom-free.

Case 6. B.S., female (40). 1946-1950—6 attacks iritis. 1950—All investigations negative, but treated with course of Lertigon* for 3 months; remained symptom-free for 12 months. 1951—Cortisone tried but not much improvement; Lertigon again; now symptom-free until 1953. 1955—Reported continuous cortisone for past 2 years with some improvement, but never symptom-free; having more Lertigon now.

Psychological trauma due to the sudden acute pain of iritis may precipitate an attack of asthma and so help in the differential diagnosis; but more often a patient has his iritis, his asthma, his migraine, or his dermatitis, as part of a system of alternating allergies, well recognized in the eczema-asthma complex, but not so well known when the manifestations form a migraine-iritis-rheumatoid arthritis syndrome.

The suggestions that some trauma is necessary before any particular tissue becomes a 'shock tissue' for an allergen to act upon leads us to consider those post-accident cases of acute cyclitis. The trauma of the localizing accident may act as a trigger for some allergic response to air-borne dusts or to drugs used in the emergency treatment; or if the lens capsule is torn by a foreign body, the surrounding tissues become sensitized by the escaping lens protein. The stage is now set for an anaphylactoid reaction in this and perhaps also in the other eye, especially if the lens protein is concerned in any operative procedure during the next few days or weeks. Case 7 illustrates such a patient:

Case 7. Male aged 38. Perforating injury of the left eye with lens puncture. No foreign-body found. Routine treatment in hospital, including penicillin locally. Discharged on 8th day. On 10th day reported at out-patients clinic with 'no pain but worried by loss of vision'. Curette evacuation of swollen lens (not whole) and A.C. wash-out. 11th day—Acute cyclitis left eye and some discomfort right eye. 12th day—Severe cyclitis both eyes: routine allergy tests: all inhalants, pollens, foods and drugs negative, uveal pigment negative, lens protein +++ (intradermal). Desensitization by graded doses of lens protein 3 hourly for 3 days. 15th day—Right eye normal in appearance and vision, left eye still slightly injected, but all discomfort gone. 17th day—Further wash-out with A.C. Lens protein disturbed without any flare-up. 23rd day—Vision right eye 6/6, left eye 6/24. 53rd day—Vision right eye 6/6, left eye 6/24.

* Lertigon is Histamine-azo-globulin of Parke Davis Ltd.

Drug Reactions

Compared with other specialities, ophthalmology tends to use few drugs: in iritis, atropine has been the constant friend of both surgeon and patient, except in the odd one in a hundred cases who shows a specific allergy to this drug. Many more than one in a hundred are hypersensitive to atropine, being able to tolerate, and be well-dilated by 1/1,000 or even 1/10,000, although 1/100 causes local stinging and burning. Those who are allergic to the atropine molecule or the tropine ring get reactions in the surrounding tissues. If these are not very intense, and atropine is necessary, one tablet of an anti-histamine given by mouth 20 minutes before each drop of atropine is applied to the eye usually keeps the condition under control for a short attack of iritis; but in severe cases some other mydriatic is necessary.

Case 8. D.L., female (58). Recurrent iritis for 8 years; attacks usually responded to treatment within 4-5 weeks. October 1954—Acute flare-up right eye; given atropine ointment within 12 hours; red, irritating eye with oedema of surrounding tissues and eczema of 2/3rds of face; allergy tests (intradermal): atropine +++, hyoscine +. N.B.—Had 12×1,000,000 units of penicillin for streptococcal pneumonia in March 1954.

In contrast to case 8 case 9 is an example of the production of an acute iritis secondary to glaucoma, presumably by the use of a new drug on this known allergic patient for treatment of his glaucoma.

Case 9. P.A., male (62). 10 a.m.—Admitted to hospital complaining of loss of vision right eye. 11 a.m.—Diagnosed by two senior ophthalmologists as glaucoma without secondary iritis and given eserine drops. 3 p.m.—Intensive pain and smarting and much oedema of lids; acute iritis on examination; intramuscular injection of 2 c.c. of anthisan given *statim* and pain and swelling controlled in 40 minutes. 3 days later—Allergy tests showed: eserine ++, prostigmin ++, pilocarpine −; glaucoma being controlled with pilocarpine as required.

Ideally every patient with a past history of allergy should be tested intradermally with any drug new to this particular patient. These drug reactions are usually of the immediate or extracellular type and show a very definite skin reaction in contrast to a control saline-test within 20 minutes, though it must be remembered that the same patient may at one time have an extracellular reaction and at a later date an intracellular one to the same drug; that is to say, his secondary allergy may be controlled by anti-histamines or may need cortisone.

Are more patients showing allergic manifestations today than 20 years ago? The answer is 'yes' and the reason seems to be not the number of new drugs and foods used daily in this and in other countries but the types of the drugs. Some antibiotics are now known to act as sensitizers, and create or intensify the particular biochemical metabolic upset which is known as the 'allergic state' and is sometimes coupled with that certain hostile outlook on life so often referred to in the study of psychosomatic medicine.

The simplest illustration is the hay-fever patient who has his annual course of anti-pollen injections with good results and no untoward reactions, until he has penicillin

for some intercurrent infection one winter: then next spring the first minute dose of pollen solution produces an attack of hay-fever, accompanied by oedema of the lips, eyelids and occasionally glottis, and may cause a spasmodic wheeze from the chest. Perhaps some of you have patients whose iritis recurs each summer, and heals at the end of the pollen season—and only then, in spite of all your care and attention. Then you would be wise to test for pollen allergy and, if the result is positive, take a careful history of contact with antibiotics, weed-killers and dyes, such as those containing a p-phenylene-diamine group (see case 8).

SUMMARY

Iritis is presented as an occasional manifestation of allergy. It may be of either the extracellular or the intracellular type and may be due to pollen, inhalants, foods, drugs or toxin (bacterial or virus). Both anti-histamines and cortisone have a place in the treatment of the acute stage but take no part in the ultimate prophylactic treatment of recurrent iritis of allergic origin.

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DISCIFORM KERATITIS AND CORTISONE*

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The name disciform keratitis was coined by Fuchs in 1901. It is more non-committal and less sibilant than the term abscessus siccus which it replaced. It is not a disease *sui generis*, but a description of any non-suppurative lesion of the corneal stroma characterized by disc-shaped swelling and opacity. It may occur after trauma, as well as in association with virus infections such as vaccinia and varicella. It is, however, its frequent association with herpes-febrilis infection which is perhaps of greatest interest.

The mechanics of the corneal swelling in disciform keratitis is not clear. Braley[1] postulates that it represents a hypersensitivity to herpes and that the herpes virus, acting as an antigen, combines with the local and circulating antibodies to produce a hypersensitivity reaction. The evidence for this view does not appear to be conclusive.

In the few cases of disciform keratitis which I have treated recently, I have found cortisone to be effective in clearing the interstitial corneal opacity, yet I am dubious about using corticosteroids freely in cases of this condition in which I suspect a herpetic origin.

In the first place one must take cognizance of the known dangers of cortisone in the other clinical types of herpes infection of the cornea. Thus Thygeson[2] states that before the advent of cortisone perforation and hypopyon were unheard of complications in herpes corneae. He reports that he has seen 2 cortisone-treated cases with hypopyon and knows of 3 with corneal perforation. He puts forward the view that only harm can result from the widespread use of cortisone in herpetic infections. Braley states that cortisone disturbs the local immunity in the cornea to the herpes virus. He quotes H. L. Ormsby in stating that cortisone applied to the cornea at the beginning of a dendritic ulcer will spread the lesion to the entire cornea. On theoretical grounds, therefore, it would seem that caution is necessary in the exhibition of cortisone in the herpetic type of disciform keratitis.

Secondly, while it is accepted that some cases of disciform keratitis respond well to cortisone, it must also be recognized that other cases become worse under adrenal corticosteroid treatment. Thus Hogan *et al.*[3] record that of 8 cases treated with hydrocortisone 2 gave a good response while the other 6 were worse. Another 8 cases treated with cortisone gave an excellent response in 5, but 3 were worse after treatment.

Case Report: A White South African male complained of defective vision in one eye. Examination showed a round swelling of the parenchyma in the optical zone of the cornea. The posterior surface of the cornea bulged toward the anterior chamber. Keratic precipitates were present. Vascularization of the cornea was absent. With topical cortisone the corneal opacity and swelling resolved rapidly, but the patient developed a dendritic ulcer of the cornea. The cortisone was stopped and the ulcer was carbolized and aureomycin ophthalmic ointment prescribed. The ulcer healed within about 48 hours.

CONCLUSION AND SUMMARY

Many cases of disciform keratitis are of herpetic origin. In view of the known dangers of adrenal corticosteroids in other herpetic infections of the cornea and the fact that cases of disciform keratitis may deteriorate on this form of treatment, corticosteroids should be used with great caution in this condition. Subconjunctival injections of the drug, in forming a local tissue-depot of the drug which cannot readily be withdrawn, are potentially dangerous.

A case of keratitis treated with topical cortisone is described.

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* A paper presented at the South African Medical Congress, Pretoria, October 1955.

CUTANEOUS MANIFESTATIONS OF TUBERCULOSIS IN THE WESTERN CAPE

A REVIEW OF THE LAST DECADE*

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Skin tuberculosis has been chosen as the subject of this paper for two main reasons—firstly because a fair number of cases of one form or another is seen at the Cape, and secondly because the treatment of tuberculosis has undergone a revolutionary change in the last 10 years. The time therefore seems opportune for a general review of the situation.

For the 10-year period ended 31 May 1955 records are available at the Groote Schuur Hospital of 50 cases of tuberculoderms; of these, 34 are lupus, 6 scrofuloderma, 2 tuberculosis verrucosa cutis, 3 papulonecrotic tuberculid, 2 erythema induratum of Bazin, 2 rosacea-like tuberculid of Lewandowsky and 1 lupus miliaris disseminatus faciei. There have been no primary cutaneous complexes; these are rarely diagnosed in South Africa.[1]

As regards erythema nodosum, many cases are seen in dermatological, medical and paediatric out-patient sessions, but few are admitted. Unfortunately, records are obtainable only of warded cases; of these, there have recently been 4 patients suffering from tuberculosis who have presented with erythema nodosum, all of them children with demonstrable primary lung complexes.

It is remarkable that, amongst all the cases of florid, active pulmonary tuberculosis seen in their hundreds at the Cape institutions, the medical officers seldom see an eruption directly attributable to infection of the skin by the tubercle bacillus. Cipollaro[2] notes a similar state of affairs in the United States.

In the last 10 years 20,133 cases of tuberculosis have been notified to the Medical Officer of Health in Cape Town. Presumably, most cases of skin tuberculosis are seen at the Groote Schuur Hospital, rather than at the tuberculosis clinics and sanitoria, so that our figures are fairly representative of the total number of skin cases seeking advice.

The distribution of the 50 cases at the hospital was as follows: Cape Town (30), Strand (2), Kraaifontein (1), Philadelphia (1), Worcester (1), Genadendal (2), Bredasdorp (2), Tulbagh (1), Moorreesburg (1), Citrusdal (2), Van Rhynsdorp (1), Blanco (1), George (1), Prince Albert (1), Richmond (1), Port Elizabeth (1), Transkei (1). Most of the local patients live in the poorer parts of the Cape Peninsula and its environs.

It will be seen from these figures that tuberculosis of the skin is a relatively uncommon form in the Western Cape and is by no means the problem that it still remains in European countries,[3] despite the very high incidence of tuberculosis in general in South Africa. Perhaps the greater amount of sunlight is a factor in protecting the skin from invasion by the tubercle bacillus. Nevertheless, it is a problem that has to be tackled, particularly amongst the non-European lower income groups. Tuberculoderms are rare in European South Africans, only 5 cases having been seen at the Groote Schuur Hospital in the last decade, 3 of lupus and 2 of rosacea-like tuberculid. The figures and distribution of our cases are indicative of the important part played by bad socio-economic conditions in the etiology of cutaneous tuberculosis.

An attempt is being made to investigate the family backgrounds of all our cases; so far, very few have revealed tuberculosis in other members of the families.

The diagnosis of many of our cases has been confirmed by biopsies at the outset, and 'test of cure' biopsies are performed towards the end of the course of treatment. Acid-fast bacilli are very rarely seen in our histological sections. Tuberculin tests, erythocyte sedimentation rates and chest X-rays have been done as a routine measure in the last few years. Only 9 of the tuberculoderms were associated with active pulmonary tuberculosis (3 lupus, 2 erythema induratum, 2 scrofuloderma, 1 papulo-necrotic tuberculid and 1 tuberculosis verrucosa cutis). Five other cases showed calcified hilar glands. Of the 28 cases who were tuberculin tested, only 3 were negative; 1 was a Native boy with lupus and the other 2 were adult Europeans, the one a woman suffering from a rosacea-like tuberculid and the other a man with lupus-like lesions on the thigh, histologically found to be tuberculous. Of the 29 cases in whom an erythrocyte sedimentation rate was done, it was found to be raised in 18, of whom 6 had active pulmonary tuberculosis. It has been noted that the purely cutaneous cases usually show a gradual decline of the erythrocyte sedimentation rate towards normal as the lesions respond to treatment.

THE TREATMENT OF TUBERCULOSIS OF THE SKIN

Now that there are 4 powerful anti-tuberculous chemotherapeutic agents—calciferol, streptomycin, isonicotinic acid hydrazide and para-aminosalicylic acid—cutaneous tuberculosis can be regarded as curable in a reasonable period of time. This is a far cry from the sad old days when an adolescent with lupus would be doomed to a *via dolorosa* of clinics and unsatisfactory treatment, with gradual progression of the scarring, destructive process. Perhaps this is an exaggeration, because good results *were* obtained in a proportion of cases. Before the new era of treatment of lupus in the early nineteen forties, therapy aimed at improving the general condition and building up the body's resistance to infection by means of good food with supplementary vitamins, iron and general ultra-violet light baths. Even today we should continue to employ these general

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supportive measures, because we are, after all, dealing with a tuberculous state. Certain systemic modalities were used with varying success; for example, gold injections, thyroid extract, and tuberculin, which produced a marked local reaction in the lupus. Local destructive measures were frequently used, amongst which were surgical excision (which still has a place in very early lupus), scraping, arsenic, trichloracetic acid, salicylic acid, mercury, potassium permanganate, nascent iodine, lactic acid, acid nitrate of mercury, phenylethyl hydnocarpate and pyrogallic acid (in fact, the old story of the number of suggested remedies being almost directly proportional to the incurability of the disease in question). X-ray therapy was used at one time but fell into great disrepute because of the danger of malignant changes at the site; these, of course, can occur spontaneously in untreated lupus and, whereas epithelioma is the usual complication, the one case in our series which underwent degeneration developed a basal-cell carcinoma which had to be widely excised; she had had no radiotherapy to the lupus but had had years of ultra-violet light locally. Rodent ulcers are exceedingly rare in the pigmented races in this country, and it is possible that the prolonged, intensive exposure to actinic light might have been a factor in producing the basal-cell carcinoma in this Coloured woman.

Niels R. Finsen's name will forever be associated with the early cures of the hitherto incurable lupus. It was in 1895 that he first used his famous arc lamp in the treatment of lupus. A marked inflammatory reaction in the lupus was aimed at, to bring about destruction of the 'apple jelly' nodules, but only recently was it realized that the local formation of vitamin D in the tissues was probably partly responsible for the good results.

As Michelson says in his foreword to Riehl and Köpf's book: 'The therapy of lupus vulgaris was essentially a physical one until recent times when now the attack is largely chemical.[4]

Calciferol.

In 1943-4, Charpy in France published his excellent results of vitamin D2 in the treatment of lupus.[5-7] At about the same time, though independently, Fanielle in Belgium[8] and Dowling and Prosser Thomas in England[9 11] were using calciferol in large doses. This was an epoch-making step forward in the therapy of cutaneous tuberculosis. Calciferol has no action on the tubercle bacillus *in vitro* and is thought to act only on the tissues, stimulating connective-tissue proliferation and causing disintegration of the granulomatous deposits.[12]

Vitamin D2 is formed by the irradiation of its precursor, ergosterol, by ultra-violet light.[13] It has profound effect on calcium-phosphorus metabolism, influencing the absorption of calcium by reason of its action on the intestinal mucosa. In the treatment of cutaneous tuberculosis, calciferol is given in massive doses ranging from 50,000 to 300,000 international units a day. It is made up in tablet form, each tablet consisting of 50,000 units. The effective therapeutic dose approaches the limit of tolerance. Over-dosage produces hypercalcaemia, although symptoms may appear without any rise in serum calcium. There is, too, a rise in serum inorganic-phosphorus and blood urea, and a fall in plasma phosphatase. The marked withdrawal of calcium from the skeleton causes an increased urinary output of calcium and may eventually lead to osteitis fibrosa cystica and metastatic calcification in the soft tissues.

The earliest toxic symptom is, paradoxically, an unusual feeling of increased well-being which is, however, soon followed by anorexia, nausea, vomiting, headache, thirst, lassitude, muscular atony, constipation or diarrhoea with bloody stools, acute abdominal pain, psychic depression, polyuria with albuminuria and rapid loss of weight. An excess of calcium and phosphorus in the diet sensitizes the patient to intoxication by vitamin D; it is therefore inadvisable to supplement the calcium and phosphorus intake. Vitamin D is slowly excreted and has an accumulative effect; the toxic symptoms are, however, reversible on discontinuing the drug, although evidence of renal damage may persist for a year or more after cessation of treatment, particularly in adults. The erythrocyte sedimentation rate rises with approaching toxicity.

With these toxic effects in mind, there are certain routine tests which are regularly performed on patients on prolonged courses of calciferol, the most important of which are weekly estimations of serum calcium, inorganic phosphorus and urea, together with examinations of the urine for albumin and excess calcium. The patient must be weighed once a week and instructed to report immediately any of the symptoms just described. It is considered highly inadvisable to administer calciferol to patients suffering from active pulmonary tuberculosis. If metastatic calcification is suspected, straight X-rays of chest and abdomen will reveal this. If the serum-calcium level reaches 12 mg.% the dose of calciferol should be reduced or discontinued altogether. In view of the very toxic nature of this drug hospitalization of the patient for a few weeks or months is highly desirable.

Streptomycin.

This antibiotic is derived from the Streptomyces sub-group of the soil Actinomycetes. It first appeared in 1944 and has proved itself to be of inestimable value in the treatment of all forms of tuberculosis. In 1947 streptomycin was given, in combination with promizole, by O'Leary and others[14] to 15 cases of skin tuberculosis without any great success. In 1948 it was first used by Cornbleet[15] in combination with calciferol in the treatment of lupus vulgaris and was thought to enhance the effect of the latter in a synergistic way; Cornbleet suggested that calciferol sensitized the tubercle bacillus to the action of streptomycin. Charpy, Dowling and Prosser Thomas all observed residual nodules after prolonged treatment with vitamin D2. Cornbleet found that, after a 6-9 weeks' course of combined streptomycin and calciferol, the scars were thin and atrophic and contained no active nodules.

There are two forms of this antibiotic, dihydrostreptomycin and streptomycin; the former may have a permanent deleterious effect on the auditory nerve and the latter may damage the vestibule. It has been found that a combination of the two in equal parts is far less toxic than either used alone. They are bacteriostatic, not bac-

tericidal, and the tubercle bacillus soon develops resistance to them if they are used alone. In dermatological practice, the combination of dihydrostreptomycin and streptomycin is used, invariably in conjunction with calciferol, isoniazid or para-aminosalicylic acid.

As with all the other antibiotics, toxi-allergic eruptions are sometimes seen, usually of the erytheme multiforme or urticarial types, but occasionally morbilliform, scarlatiniform or haemorrhagic. True exfoliative dermatitis has been known to supervene. Most of these eruptions appear within the first 10 days of treatment and are easily controlled by antihistaminics. Stomatitis and generalized pruritus may be distressing symptoms. The most serious side-effects of these two antibiotics are those resulting from damage to the auditory nerve and vestibule, leading to deafness, tinnitus and vertigo. Permanent deafness as a rule only results from intrathecal use of dihydrostreptomycin for tuberculous meningitis,[16] but has recently been described after prolonged intramuscular use.

Isonicotinic Acid Hydrazide (*Isoniazid, INH*)

This drug made its appearance early in 1952. It is a coal-tar compound and is not really a newcomer, its formula having been discovered in Prague 40 years ago. It was discarded and forgotten until, in 1945, a French scientist observed that niacinamide, in the vitamin-B constellation, inhibited the growth of the tubercle bacillus in animals. Chemists of the Squibb and Hoffman-La Roche laboratories, working independently, after 5 years' research isolated isonicotinic acid hydrazide which could be cheaply produced and was effective, when taken by mouth, in inhibiting the *Mycobacterium tuberculosis*. It is well known that *in vitro* the bovine bacillus is insensitive to INH whereas, in actual practice, lesions produced by these organisms respond very well; this would seem to be an indication of the unreliability of laboratory procedures when attempting to assay the antibacterial efficacy of a drug.[17] Unfortunately, *in vivo* the organism does very soon become resistant to INH, but this can be combated by giving streptomycin or PAS at the same time.

Isoniazid is given by mouth in tablet form, each tablet consisting of 50 mg. The dose is 3-6 mg. per kg. of body-weight per day, the usual amount given to an adult being 200-400 mg. per day in 3 or 4 divided doses.

The toxic effects of INH simulate vitamin deficiencies and are largely the result of irritation of the central and automatic nervous sytems. Side-effects are rare, particularly in children, and tend to occur during the first 3 weeks of treatment; they usually disappear in spite of persistent administration of the drug. The symptoms referable to the nervous system consist of muscular twitchings, hyperreflexia, vertigo, dryness of the mouth, constipation, hypertonia of the bladder sphincter, euphoria, mental excitability and insomnia. Peripheral neuritis with marked sensory distubance has been described by Linton, Rabinowitz and Olie at Springkell[18] and by others elsewhere. Anorexia, loss of weight and jaundice due to toxic hepatitis are occasionally met with, and transient blood-changes in the form of eosinophilia and mild anaemia have been observed.

The vitamin-B group, particularly pyridoxine, is useful in counteracting many of the toxic effects of INH. It is thought that INH exerts an antivitamin effect by blocking the action of nicotinic acid and pyridoxine, or that it depletes the tissues of pyridoxine.[19] An acute pellagrinous picture has been described by McConnell and Cheetham.[20]

Para-aminosalicylic Acid (*PAS*)

This drug has only a very slight bacteriostatic effect on the tubercle bacillus but is of immense value in reducing the resistance of this organism to streptomycin and INH, and is now used almost exclusively as an adjunct to these latter in the treatment of tuberculosis It is given by mouth in tablet form, each tablet consisting of ½ g. of PAS, in 4-hourly divided doses totalling 12-20 g. a day. Children are given 125 mg. per kg. of body-weight, in orange juice, 6-hourly.

PAS is apt to cause the same toxic symptoms as the other salicylates, namely, drug fever, rashes, tinnitus, nausea, vomiting, diarrhoea, jaundice and hypokalaemia with paralyses and cardiac arrhythmias. Lymphadenopathy, hypoprothrombinaemia, albuminuria, haematuria and even anuria have all been reported. It should be mentioned, in passing, that PAS reduces Benedict's reagent; this is obviously of importance when treating patients who are suffering from diabetes as well as tuberculosis.

EXPERIENCES AT THE GROOTE SCHUUR HOSPITAL

In 1944 we first used calciferol for lupus vulgaris at Groote Schuur Hospital. Nine years ago, I read a paper at the Durban Congress giving our results in the few cases we had treated. We were very pleasantly surprised by the comparatively good outcome, although it was still a matter of half a year or more before any striking improvement took place (Figs. 1 and 2).

In 1949 we first combined streptomycin with calciferol

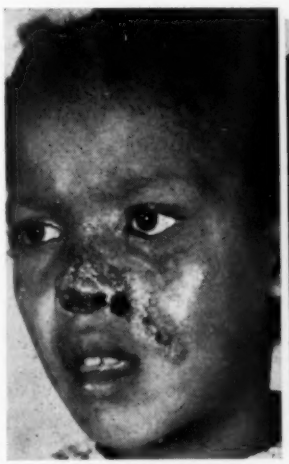

Fig. 1.

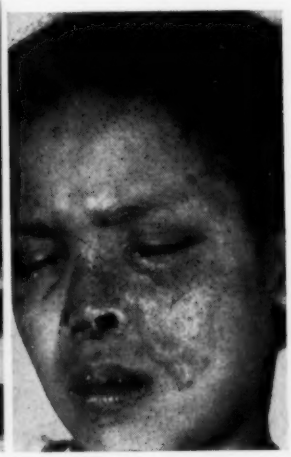

Fig. 2.

Fig. 1. The first case treated with calciferol at Groote Schuur Hospital. Before treatment.
Fig. 2. Same case as Fig. 1 after 5 months on calciferol.

and were even more gratified by the distinctly quicker progress of our cases.

In 1952 we decided to try out the new isonicotinic acid hydrazide. We used it alone at first and were astounded by the remarkably rapid healing of a very severe case of scrofuloderma within 3 weeks (Figs. 3 and 4).

Since then we have used varying combinations of INH, streptomycin and PAS with, on the whole, very satisfactory results in most cases, although some seem to become resistant to these 3 after a while; under these circumstances, we use calciferol, which invariably has the effect of finally clearing up the skin infection.

Table I gives more detailed information about the 4 drugs.

We have formed certain general impressions about the chemotherapy of the tuberculoderms.

The earlier cases on INH cleared with astonishing rapidity, so much so that we found ourselves saying that INH could do in weeks what it took calciferol months to accomplish. We have, however, had to revise that opinion in the last 2 years, and it seems as though the tubercle bacillus has become fairly resistant to this newest and most dramatic of the chemo-therapeutic agents at our disposal, a situation perhaps comparable with the resistance to penicillin that has been acquired by the staphylococcus. We, therefore, remain loyal to calciferol, which is still the great stand-by when INH, streptomycin and PAS let us down.

The true tuberculous infections, lupus and scrofuloderma, tend to respond better to chemotherapy than do the so-called tuberculids, particularly the more anergic forms like lupus miliaris disseminatus faciei. Michelson[21] states that it has been the experience of the German dermatologists that lupus miliaris does not respond to INH, somewhat strengthening the view that, in spite of its histology, it may not be tuberculous. Our case showed very little improvement while on INH and streptomycin, but healed on calciferol.

At this point, sarcoid might be mentioned. This is not the time to go into the whole vexed question of sarcoidosis, but one might just say, in passing, that two cases of nodular sarcoid cleared up on calciferol without any of the unpleasant sequelae so often manifested in this disease.

We have been most fortunate in the rarity of side-effects and symptoms of toxicity in our patients. Ten of our 50 cases exhibited side-effects to calciferol and 2 of these 10 to streptomycin as well. The calciferol intolerance revealed itself as hypercalcaemia, excess urinary calcium, albuminuria, dryness of the mouth with fatigue, nausea and anorexia, acute abdominal pain, constipation and headaches. One patient who showed signs of intolerance to streptomycin complained of clicking and deafness in the ears and the other had an anaphylactic crisis. They all recovered within a few days of discontinuing the drugs and, in some instances, were able to tolerate later courses very well.

Local ultra-violet light therapy is given throughout the course of chemotherapy.

Fig. 3.

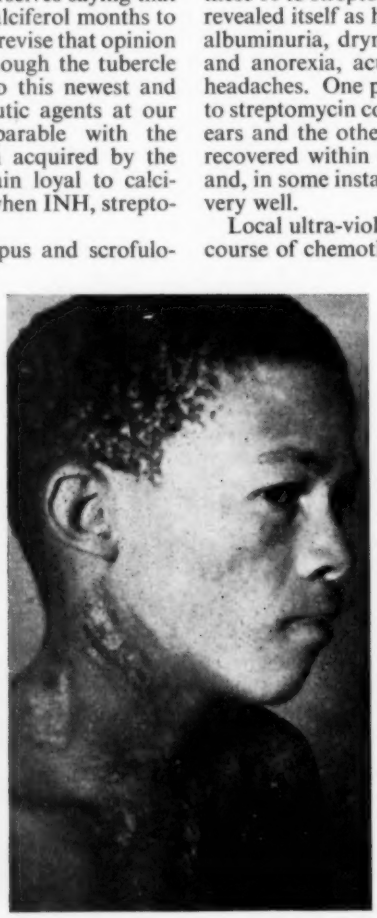

Fig. 4.

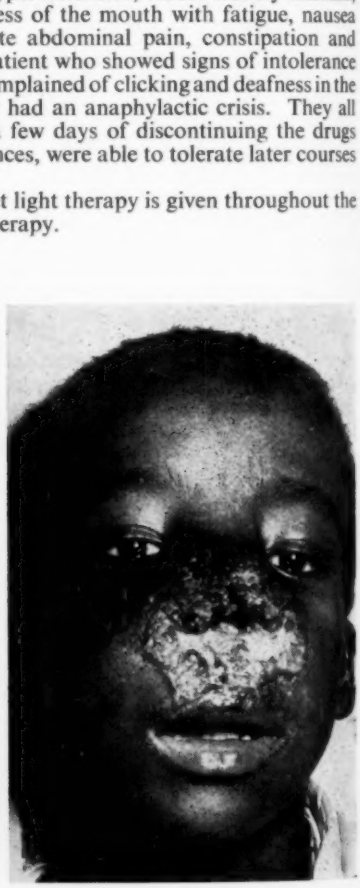

Fig. 5.

Fig. 3. The first case treated with INH at Groote Schuur Hospital. Before treatment.
Fig. 4. Same case as Fig. 3 after 3 weeks on INH.
Fig. 5. Neglected lupus.

The great advances in plastic surgery have made possible the repair of ravaged faces, now that dermatologists are able to eradicate the active tuberculous process before destruction of tissue has progressed too far. Before operating it is essential to wait, for at least 6 months after apparent cure, to be certain that no new lupus nodules are forming.

CONCLUSION

I can best summarize my talk by the remark that, nowadays, when a patient with lupus comes in, our former sense of frustration and commiseration has been replaced by a feeling of joy that yet another sufferer seeks easily-procured relief from one of the most distressing of maladies.

All forms of cutaneous tuberculosis are compulsorily notifiable in South Africa. Perhaps because of their non-infectious nature, this is not realized by all practitioners.

BCG vaccination is worthy of mention. It is not as yet being practised on any large scale at the Cape, but, when it is, we may expect to see the occasional case of lupus developing at the site of innoculation.[22-25]

I should like to have the news of the great efficacy of the modern therapy of lupus blazoned to all the corners of the Union of South Africa, so that the sufferings of the people in the locations and slums may be alleviated by this treatment, which is simple and may readily be carried out by the District Surgeons in collaboration with the District Nurses. Hospitalization is by no means essential when streptomycin, INH and PAS are used, and complicated laboratory procedures may well be dispensed with. Fig. 5 illustrates a tragedy that to-day is a disgrace in a civilized country.

I should like to thank Dr. N. H. G. Cloete, Superintendent of Groote Schuur Hospital, Cape Town, and Dr. R. Lang, Head of the Department of Dermatology, for permission to publish this paper. Thanks are due, too, to Mr. B. Todt for the photographs.

TABLE I

VITAMIN D2 (CALCIFEROL)

Tablets: 50,000 i.u. each. Oral.
Dose: 50,000 to 300,000 i.u. daily.
Contra-indications: Pulmonary tuberculosis, arteriosclerosis, kidney disease.
Toxicity counteracted by vitamin A (partly).
Toxic Effects mainly due to altered calcium-phosphorus metabolism.

Symptoms and Signs of Toxicity

Early increased feeling of well-being.
Anorexia, nausea, vomiting, constipation or bloody diarrhoea
Acute abdominal pain.
Pyrexia.
Rapid loss of weight.
Dry mouth, thirst.
Polyuria, albuminuria, increased urinary Ca.
Hypertension.
Headache, psychic depression, hazy memory, dizziness, mandibular neuralgia, tender gums and teeth. Paraesthesia, peripheral neuritis, optic atrophy. Retinal vessels affected.
Muscular and articular pains.
Libido increased or diminished.
Leucocytosis. FSR rises with approaching toxicity.
Increased tuberculin allergy during treatment is a favourable sign.
Metastatic calcification (if in placenta, foetal death may occur).
Rarefaction of bones.
Increased serum calcium, inorganic phosphorus and blood urea.

STREPTOMYCIN

Intra-muscular injection.
Dose: 1 g. daily at first. Children: 0·01 g./kg body-weight/day.
Contra-indications: None, unless known allergic sensitivity is present.
Toxicity counteracted by antihistaminics (partly).
Toxic Effects: Allergic and aural.

Symptoms and Signs of Toxicity

Tinnitus, vertigo, deafness.
Eruptions: Urticarial, erythema multiforme, morbilliform, scarlatiniform, haemorrhagic, exfoliative dermatitis.
Stomatitis.
Pruritus.
Anaphylaxis.
Tight feeling around the mouth.
(Contact dermatitis in personnel).

ISONICOTINIC ACID HYDRAZIDE (INH)

Tablets: 50 or 100 mg. each. Oral.
Dose: 3-6 mg./kg body-weight, usually 300-400 mg. daily. Children: 6 mg./kg.
Contra-indications: None. Avoid concomitant administration of CNS stimulants (ephedrine, belladonna, adernaline).
Toxicity counteracted by: Pyridoxine.
Toxic Effects mainly due to stimulation of central and autonomic nervous systems.

Symptoms and Signs of Toxicity

Anorexia (or enormous appetite), dry mouth, heartburn, nausea, vomiting, constipation or diarrhoea, abdominal discomfort.
Loss of weight.
Jaundice.
Transient flushing of face.
Peripheral neuritis. Paraesthesia. Burning feet. Muscular twitching. Motor restlessness. Ataxia. Coarse tremors. Hyperreflexia. Transitory Babinski sign. Bladder sphincter hypertonia. Retention.
Transient albuminuria.
Vertigo. Euphoria. Insomnia. Mental excitability and irritability. Fatigue. Loss of memory. Meningism. Increased susceptibility to convulsions in epiletics. Confusional psychosis. Headache. Stupor. Drowsiness.
Hypotension. Palpitations. Angina in arteriosclerotics.
Death in respiratory failure.
Anaemia. Eosinophilia. Increased coagulation time. Increased bleeding tendencies. Agranulocytosis.
Eruptions: Eczematous. Generalized desquamation. Herpes zoster.
Pellagra.
(Contact dermatitis in personnel.)

PARA-AMINOSALICYLIC ACID (PAS)

Tablets: ½ g. each. Oral.
Dose: Adults 12-20 g. daily. Children ¼ g./kg. daily in orange juice.
Contra-indications: None.
Toxicity counteracted by: Sod. bicarb. for gastric-intestinal disturbances.
Toxic Effects similar to other salicylates.

Symptoms and Signs of Toxicity

Rashes.
Drug fever.
Nausea, vomiting, diarrhoea.
Jaundice.
Lymphadenopathy.
Hypokalaemia.
Cardiac arrhythmias.
Hypoprothrombinaemia.
Tinnitus.
Albuminuria. Haematuria. Anuria. Urine (reduces Benedict's).

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OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

VACANCY—ASSISTANT EDITOR

Applications are invited from medical practitioners for the post of Assistant Editor in the service of the Medical Association of South Africa at its Head Office in Cape Town.

The salary scale attaching to the post is £1,250×50—1,750 per annum, plus an annual cost-of-living allowance of £176 for single men and £352 for married men. The commencing salary will be determined according to journalistic experience.

The successful applicant must contribute to the Association's Superannuation Fund. He will also be expected to assume duty as soon as possible after appointment.

Applications must reach the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town, on or before 2 March 1956.

A. H. Tonkin
Secretary

Medical House
Cape Town
23 December 1955

VAKATURE—ASSISTENT-REDAKTEUR

Aansoeke word van geneeshere ingewag vir die betrekking van Assistent-Redakteur in diens van die Mediese Vereniging van Suid-Afrika, by die Hoofkantoor te Kaapstad.

Die salarisskaal aan die pos verbonde is £1,250×50—1,750 per jaar, plus 'n jaarlikse duurtetoeslag van £176 vir 'n ongetroude en £352 vir 'n getroude man. Die aanvangssalaris sal volgens joernalistieke ondervinding bepaal word.

Die suksesvolle kandidaat moet by die Vereniging se pensioenskema aansluit. Hy sal ook verwag word om so spoedig moontlik na aanstelling diens te aanvaar.

Aansoeke moet die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, bereik vóór of op 2 Maart 1956.

A. H. Tonkin
Sekretaris

Mediese Huis
Kaapstad
23 Desember 1955

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Distaquaine V Tablets. British Drug Houses are now distributing 'Distaquaine' V tablets in South Africa, manufactured by The Distillers Company (Bio-chemicals) Ltd. They submit the following statement:

'Distaquaine' V tablets consist of phenoxymethylpenicillin, a new form of penicillin which is stable in an acid medium and therefore requires no added buffer to prevent its destruction by the gastric contents. As a result of its acid stability, 'Distaquaine' V reaches the duodenum unchanged and there its absorption is immediate and uniform, giving reliable and consistent blood levels which are approximately twice as high as those resulting from an equivalent dose of penicillin G by mouth.

Indications. All diseases due to penicillin-sensitive organisms except in cases where oral medication is contra-indicated, e.g. in the presence of severe vomiting. In such cases, after an initial period of parenteral treatment, continuation with oral 'Distaquaine' V will frequently be possible.

Dosage. Each 'Distaquaine' V tablet contains 60 mg. of phenoxymethylpenicillin, which is the equivalent of 100,000 units. The average adult dose is 1 tablet every 4 hours, but in severe infections, or where a high blood level is required, an increased dosage will be necessary and will depend upon the clinical circumstances in each case. For example in one case of ulcerative endocraditis, 20 tablets were given 4-hourly over a period of several weeks without any untoward effects. To avoid disturbing the patient's sleep the last dose at night may be doubled in all but the most severe infections. To ensure maximum absorption 'Distaquaine' V tablets should be administered ½ hour before and not less than 3 hours after meals. In no case should the total daily dosage be less than 360 mg. for an adult, 180 mg. for a child and 90 mg. for an infant, in order to avoid producing resistant strains of bacteria. The tablets should not be sucked or chewed but should be swallowed quickly with a glass of water.

Tolerance. 'Distaquaine' V tablets are particularly well tolerated. Allergic reactions, when they occur, are milder than those following parenteral penicillin, Diarrhoea may occasionally appear, but is never severe, and will usually stop spontaneously within 24-48 hours. There is no gross disturbance of the intestinal flora.

Detailed literature is available on request from British Drug Houses, P.O. Box 372, Johannesburg.

Instrument for handling of surgical blades. Bard-Parker have devised a new instrument to facilitate the taking on or off from handles of detachable surgical blades. It is much easier than fingers or any other forceps, besides saving valuable time in the operating theatre. Like all Bard-Parker products, it is a precision job and is of stainless steel. The agents are Gurr Surgical Instruments (Pty.) Ltd., of P.O. Box 1562, Johannesburg, who supply this notice and advise us that the price is 32s. each.

Foot Pedal Dispensers designed to facilitate the handling of liquid soaps, in the operating theatre and elsewhere, manufactured in South Africa, are now available under the name 'Gill Dispenser' They are built of non-corrosive materials and are portable so that they can be placed in any desired position; and the quantity of soap is controlled by an easily operated foot pedal.

A glass-framed chart of instructions is supplied with the dispenser. Full details are obtainable from Messrs. Selected Pharmaceuticals Ltd., 81 de Villiers Street, Johannesburg.

REPORT OF SUB-COMMITTEE TO ENQUIRE INTO MEDICAL EDUCATION AND INTERNSHIPS*

Members of the Sub-Committee: Cape Province. Mr. M. Cole Rous† (Chairman and Convener), Mr. T. B. McMurray (Hon. Secretary), Dr. J. R. E. Lee, Mr. J. D. de B. Joubert (co-opted); *Transvaal.* Dr. J. H. Struthers, Dr. F. Ziady and Dr. M. C. Segal; *Natal.* Dr. A. B. Taylor; *O.F.S.* Dr. R. Theron and Dr. J. S. Visser.

This Report consists of two sections: (A) Medical Education and (B) Internships.

The Cape Section of the Sub-Committee decided that it was necessary in the first instance to make a study of the thought and work that has already been put into this problem. To this end the following literature was acquired and carefully studied:

1. *First World Conference on Medical Education.* London, 1953. Report by the Editor of the Proceedings, Dr. Hugh Clegg. 804 pages.
2. *General Practice and the Training of the General Practitioner.* Report of a Committee of the Association (B.M.A.), 1950. 88 pages.
3. *Psychiatry and Medical Education—Report of the* 1951 *Conference on Psychiatric Education*, under the auspices of the American Psychiatric Association. 164 pages.
4. *Psychiatry in a General Hospital.* G. A. Elliott, M.D., F.R.C.P., Professor of Medicine, University of the Witwatersrand. S. Afr. Med. J., 3 July 1954 (**28,** 561).
5. *Trends in Medical Education in South Africa. Idem. Ibid.*, 7 May 1955 (**29,** 432).
6. *A Physician Views Psychotherapy. Idem. Ibid.*, 20 November 1954 (**28,** 981).
7. *The Training of Students in General Practice. Idem. Ibid.*, 5 February 1955 (**29,** 134).

It was further decided that Mr. T. B. McMurray would undertake the extensive and onerous work of collecting information about hospital posts in the Union of South Africa. In the light of this data the problem of arranging and determining internships can be studied.

A. MEDICAL EDUCATION

M. COLE ROUS, M.B., CH.B., F.R.C.S.†
Cape Town

The term Medical Education covers both undergraduate and post-graduate teaching of medicine. The Cape members of the Sub-Committee have found the problem so complex and difficult that only the undergraduate aspect of the question has been tackled in the first instance.

After months of study, many meetings and numerous discussions, we feel that we are only just beginning to come to terms with the broad outlines of this problem and now present an interim draft report on Undergraduate Medical Education.

The Undergraduate Medical Curriculum

Informed opinion, from all parts of the civilized world, seems almost unanimous in condemning medical curricula as unsatisfactory.

The members of your Sub-Committee would like it to be clearly understood that the material on which this report is based was gathered from a study of authoritative literature and not from the study of medical curricula of South Africa. Such criticisms as are made in this report may, or may not, apply to our medical schools but are not directly aimed at them because we have made no study of them. We are aware that at least some of the medical schools of South Africa are striving to change their curricula in directions adumbrated in this report.

Faults of the Medical Curriculum. The main faults of the medical curriculum would appear to be:

(*a*) Overcrowding of the time-table, which has an inhibiting effect on the mind of the student and leaves no time for thought.

(*b*) The training is in terms of fragments and facets of specializations by specialist teachers teaching in watertight compartments.

(*c*) The teaching is administered in terms of subjects which are separated from one another in compartments. Why he should learn them and how they can be of use to him later is left to such imagination as the student may possess. The interest and motivation to study would be created by the surgeon, physician and pathologist taking appropriate and applicable material into the departments of Anatomy and Physiology and teaching in cooperation and harmony with the anatomist and physiologist. But this is not in terms of the traditional curriculum. The subject is taught in pure culture and truncated by 'stop examinations'. The implication is that having passed the 'stop examination', the student may drop the subject with a sigh of relief. The application of that subject in later years, necessary to the proper understanding of the contemporary subject, is very haphazard.

(*d*) Inadequate training in important aspects of general practice.

(*e*) Inadequate and inappropriate training in psychiatry. In terms of the traditional curriculum students are taught to name the major incurable psychoses in institutions far removed from the general hospital. They are not taught the important technique of psychiatric interview nor are they made to realize the essential unity of mind and body.

(*f*) General type of teaching. The teaching in most medical schools has been done by specialists who become ever more specialized. The emphasis has been on knowledge, detail and memory rather than on general principles and a logical approach to problems.

(*g*) Attitude towards students:

The Teaching Staff: Doubtless there have been many exceptions, but all too often the attitude towards the student is that he is there as a humble spectator privileged to see a great specialist in action, or to record verbatim his words of wisdom. Attentive admiration, silent and docile compliance, have in the past been the hallmarks of the model medical student.

The Nursing Staff: The attitude of the nursing staff towards medical students has been even more rugged and outspoken. Many of us have suffered under the dominant contempt of the matriarchal ward-sister whose starched scorn has made us feel that we enter her domain on extreme sufferance.

Summary. The general result of the traditional medical curriculum has been the production of a badly over-stuffed animal in whom the teachers have little confidence and who has less in himself. There is a pious hope that these manifest defects will be corrected during the year of internship.

Answer to the Problem

Prof. Guy Elliott, who has applied his exceptional talents to this question, which he has studied intensely for many years in many countries, says, 'There is no answer to this problem.' By this we understand him to mean that the problem is not immediately susceptible of solution by thought; it is a problem that must be 'lived out', not 'thought out'. He is making remarkable contributions in reorganizing his curriculum in terms of the reality of the modern situation. His knowledge and experience are too great to permit of a theoretical solution from the armchair.

Your Committee is of the opinion that the curriculum which proves satisfactory for one medical school may not necessarily be the best for another in the same country. The needs of one country may differ widely from those of another. Furthermore the future curriculum of a progressive medical school will almost certainly be flexible and will change with the changing needs of the times. For these reasons it would seem a positive gain if some general guiding principles could be deduced which would win the assent of most of those who have this complex problem at heart. As a tentative start in this direction we propose the following suggestions:

1. Simplification of the curriculum by teaching and emphasizing the general principles of the sciences that compose Medicine and ruthlessly pruning the 'factual' type of teaching.

2. Co-ordination and integration of the teaching of subjects (examples are given in Prof. Guy Elliott's papers). This requires a radical change in the teaching methods and may meet with resistance from some members of the teaching staff. It will require for its success a sincere and respectful cooperation between surgeons, physicians, pathologists, physiologists and so on.

This principle of coordination and integration is visualized as taking place (*a*) upwards, (*b*) downwards and (*c*) collaterally in the medical curriculum:

* Presented to the Federal Council of the Medical Association of South Africa and published by order of the Council.

† Now deceased.

(*a*) The teaching of a subject will continue after the 'stop examination' in that subject has been passed and certain aspects of physiology will be taught by the physiologist to the student of pathology, medicine and surgery, in cooperation with the teachers of these subjects.

(*b*) Certain aspects of advanced subjects will be introduced to students of the earlier disciplines to illuminate and create interest and motivation, without which 'learning is a weariness unto the flesh'.

(*c*) Subjects in which the main teaching falls in the same months of the curriculum can often be integrated in order that each shall illuminate and enrich the other.

3. Training in certain aspects of general practice by and with general practitioners. This in itself constitutes a big problem, which is being vigorously tackled in different ways in different parts of the world.

4. Psychiatric training in the general hospital to illustrate the psychological disturbances that so frequently occur in patients suffering from organic illness and to emphasize the essential unity of mind and body.

5. There should be constant and sincere attempts by all teachers to train students in observation and deductive reasoning; in recognizing and defining problems; and in the use of the library and in lucidity of communication. In short, it has become an urgent necessity to train minds rather than attempt to turn the students into repositories of knowledge.

6. Every medical student should be treated with the respect and consideration due from a senior member of an honourable profession to his younger colleague. Under no circumstances should words and attitudes of ridicule and scorn and contempt towards the medical student be regarded as justifiable. On the part of the nursing staff a similar change of attitude towards medical students should be regarded as mandatory.

Resistance to Change

The members of your Committee were particularly impressed by the remark 'It is easier to change a graveyard than a medical curriculum.' There would appear to be a general feeling that there are heavy and powerful factors resisting change in a new direction.

A study of the literature would seem to indicate that the two most important factors that might impede progress are: (*a*) A shortage of money; (*b*) The attitudes and feelings of the teaching staff.

(*a*) It is well known that lack of funds is a constant source of embarrassment to the medical schools and hospitals in this country. The old method of teaching is cheaper than the proposed changes. In the traditional pattern the accent is on lecturing, and lecturing has been defined as a process by which knowledge is transferred from the note-book of the lecturer to the note-book of the student without passing through the brains of either. In these terms a lecturer or a professor can easily deal with a class of 80 to 120 students, and this is a lot cheaper than study-groups of 4-6 students being trained in observation, inference and exposition. It is clear that the application of the new principles will require more teachers than the old and these cost money.

(*b*) The attitude and feelings of the teaching staff. The members of your Committee were unanimous in conceding the obstructive effects of a shortage of money and were equally unanimous in disbelieving that serious obstruction could, or would, come from the teaching staff. They found themselves unable to give credence to the statements (1) that the greater resistance would come from the professors and lecturers themselves; (2) that the force of tradition would outweigh the manifest necessity for modern methods; (3) that they were blind to the fact that the ever accelerating accumulation of modern knowledge and the developments of new specialities has created a situation in which the old method, however satisfactory, in the 'old days' is now hopelessly inadequate.

(*c*) That members of the teaching staff are likely to view the suggested changes with suspicion, resentment and hostility as an unwarranted encroachment likely to create discomfort and the exposure of personality, inadequacies and defects.

(*d*) That they would prefer the old methods which permit of the defence mechanisms of isolation and varying degrees of aggression and hostility rather than venture into the new fields in which cooperation and learning are mandatory for the success of the new approach.

Resistance by the medical students is most unlikely because their minds are flexible and because these changes would bring new vitality and interest and comprehensibility into the medical course.

Resistance from the Medical Council will not occur once that body is assured that the proposed changes will produce a more efficient and better integrated doctor.

The Concept of the 'Basic Doctor'

This idea has been put forward as a concept which would give meaning and integration to the proposed changes in the new medical curriculum. The suggestion is that the medical schools should not attempt to produce general practitioners but should aim at training a basic doctor, i.e. one who is well trained in the basic principles of the medical sciences, who has been taught the art of observation and reasoning, the use of logic and libraries, and the ability to recognize and define problems and to communicate semantically.

With this basic training at graduation, he would then turn to any branch of medicine, in which he would study and gain experience before being licensed to practise. If it is towards a speciality that he turns after graduation, the requirements are already laid down by the Medical Council. Suitable requirements could be formulated and re-formulated in the light of experience for those wishing to qualify for general practice.

It is pointed out that this 'basic doctor' concept would remove the anomaly of medical schools training general practitioners who are not allowed to go from graduation to general practice.

Some of those who favour this idea have urged that its application would shorten the medical curriculum considerably; others that it would shorten it slightly and still others that it would shorten it not at all.

After giving a lot of consideration to this matter, your Committee decided that, provided the general principles that have been formulated in this Report are applied to the teaching of medicine it does not matter whether the product is called a basic doctor or regarded as a general practitioner who is not yet allowed general practice.

CONCLUSION

In studying the subject of Medical Education, the members of your Committee have been deeply impressed by the magnitude, importance and difficulty of this most complex problem, which will require generations of experience for its practical solution.

Your Committee is happy to acknowledge valuable assistance which it has received from Dr. T. B. Davie, Principal of the University of Cape Town, Prof. van den Ende, Professor of Bacteriology of the University of Cape Town, and Prof. Guy Elliott, Professor of Medicine, Witwatersrand University.

B. INTERNSHIPS

T. B. McMURRAY, M.B., M.Ch. Orth. (L'pool), F.R.C.S. (Edin.)

Cape Town

Since the meeting of Federal Council held in March 1955, we have tried to get the fullest possible information regarding the facilities for postgraduate study and the position of interns in South Africa. To this end all the hospitals have been sent questionnaires which, with a few notable exceptions we have received duly filled in.*

The hospitals have been classified into 3 main groups and some subsidiary groups:

Group I. These are teaching hospitals having a full specialist staff. They are closed hospitals.

Group II. These are hospitals having a full specialist staff together with some general practitioners but are not teaching hospitals.

Group III. These are hospitals staffed mainly by general practitioners but in a few of them there are specialists on the staff.

Information has been sought to find out how many interns are normally allotted to each hospital; secondly, how many full-time higher-grade appointments are allotted to each hospital; and, thirdly, how many part-time positions there are on the staff.

* The outstanding information has since been received and is embodied in the Tables I-IV.

These part-time positions have been further divided into specialist and general practitioner and the various specialties are shown.

Unfortunately it has been impossible to complete this work in the 6 months largely because some hospitals are tardy in sending their replies, but before the Federal Council meeting to be held in April 1956 we hope to have a full analysis of hospital jobs in South Africa.*

The Position of Interns

The numbers of successful graduates in medicine for 1953 and 1954 are shown in Table I. It will be seen that there is an average of 300 per year at present. Once the Stellenbosch Medical School and the Durban Medical School start graduating doctors, then this figure will obviously have to be altered. Provisionally, I think that the alteration may mean an addition of about 100 per year, giving a total of 400 per year. The number of interns employed at the different classes of hospitals (375) is shown in Table II.

Tables III and IV show the number of interns and medical staff for whom places are allotted at hospitals belonging to Group I and Group II, and also the number of beds contained in these hospitals. The medical staff other than interns are divided into 'higher', (i.e. non-specialist medical staff and 'specialist', and also into full-time (f) and part-time or visiting (v). The distribution of staff in the different hospital departments is shown.

Group-I Hospitals (Table III)

In these the interns number 120, the 'higher' staff 197 (196 full-time) and the 'specialist' staff 349 (108 full-time and 241 visiting). The number of beds in these hospitals is 5,545.

Group-II Hospitals (Table IV)

In these the interns number 182, the 'higher' staff 268 (206 full-time and 62 visiting) and the 'specialist' staff 449 (371 visiting and 78 full-time). The beddage is 8,984.

Group-III Hospitals (Table V)

These hospitals provide places for 69 interns. The number of 'higher' staff is 70, in addition to 470 general practitioners attending.

Special Hospitals

These include tuberculosis hospitals, convalescent homes, orthopaedic hospitals, maternity hospitals and industrial hospitals. The number of interns they require is less than 10.

TABLE I. SUCCESSFUL GRADUATES IN MEDICINE

	Witwatersrand	*Pretoria*	*Cape Town*	*Total*
1953	123	64	115	302
1954	116	68	122	306

TABLE II. NO. OF INTERNS AT HOSPITALS OF DIFFERENT CLASSES

Group I ..	120
Group II ..	182
Group III ..	69
Special hospitals	9
	380

TABLE III. STAFF IN GROUP-I HOSPITALS

	Medicine	*Surgery*	*Obstetrics*	*Gynaecology**	*Paediatrics*	*Orthopaedics*	*Urology*	*Ophthalmology*	*E.N.T.*	*Dermatology*	*Psychiatry Neurology*	*Anaesthesia*	*Radiology*	
Groote Schuur														837
Interns	10	10	5		2	3	2	2	1	1				36
Higher (V)														
Higher (F)	9	10	6		1	3	1	2	2	1				35
Specialist (V)	20	13	13		8	4	6	9	3	6	5	7		94
Specialist (F)	8	3	1		3	1			1	1	1	2	8	29
Johannesburg General														1,500
Interns	15	13								1				29
Higher (V)														
Higher (F)	11		10	10	3	6	2	2	3			6	4	89
Specialist (V)	20		10		10	5	5	10	10	7		8	3	99
Specialist (F)	8		4		1	2					1	10	15	43
King Edward VIII														1,653
Interns	10	9	6			1								26
Higher (V)						1								1
Higher (F)	5	4	3			2		2	1			3		20
Specialist (V)	2	6				3								11
Specialist (F)	2	6	3					1	1	1	1	3	3	21
Pretoria														1,555
Interns	9	4	3		3	5	2					3		29
Higher (V)														
Higher (F)	14	9	5		5	7	1	2	2			7		52
Specialist (V)	8	7	5		3	6	2	2	2	1	1			37
Specialist (F)												6	9	15
Totals														5,545
Interns	44	36	14		5	9	4	2	1	2		3		120
Higher (V)						1								1
Higher (F)	39	23	24	10	9	18	4	8	8	1		16	4	196
Specialist (V)	50	26	28		21	18	13	21	15	14	6	15	3	241
Specialist (F)	18	9	8		4	3		1	2	2	3	21	35	108
	151	94	74	10	39	49	21	32	26	19	9	55	42	666

*Where not otherwise stated gynaecological staff is grouped with the obstetrical staff.

In addition the Johannesburg General Hospital returned the following staff:

Higher (F). Thoracic Surgery 1, Casualties 31.

Specialist (V). Radiology 3, Physical Medicine 3, Plastic 1, Thoracic Surgery 2, Neuro-Surgery 1, Casualties 1, Peripheral Vascular 3.

Specialist (F). Physical Medicine 1, Plastic 1.

TABLE IV, GROUP-II HOSPITALS

	Medicine	Surgery	Obstetrics	Gynaecology	Paediatrics	Orthopaedics	Urology	Ophthalmology	E.N.T.	Dermatology	Psychiatry Neurology	Anaesthesia	Radiology	Total	Beds
Addington															
Interns	5	4	3		2	2	1	1	1	1	1	1		22	720
Higher (V)	2													2	
Higher (F)	5	2	2		2	1		1	1			3		23	
Specialist (V)	6	6	2		3	3	2	2	3	1	1	3		33	
Specialist (F)													4	9	
Baragwanath															
Interns	15	15												30	1,506
Higher (V)															
Higher (F)	11	13	11		11	3	2	3	1			4		66	
Specialist (V)						1		2	1					11	
Specialist (F)	7	6	4		3	2	2	1	1	1		5	5	37	
Boksburg-Benoni															
Interns	2	2	2		1	1	1	1	1					11	362
Higher (V)					1							2		3	
Higher (F)	1	1	1									1		4	
Specialist (V)	1	1	1		1	2	1	2	1	1	1		3	16	
Specialist (F)												1		1	
Cape Town Free Dispensary															
Interns	1	1												2	
Higher (V)															
Higher (F)	2	1		1		2		2	1	3		2	1	15	
Specialist (V)															
Specialist (F)															
Conradie															
Interns	1	1		1		1								4	176
Higher (V)															
Higher (F)												1		1	
Specialist (V)	2	4		2		1	2	1	1			4	1	18	
Specialist (F)															
Coronation															
Interns	5	6												11	410
Higher (V)															
Higher (F)	6	7		3	9	1						3		29	
Specialist (V)				2	2	1	1	1	1	1	1			11	
Specialist (F)	3	3										3	2	11	
Discoverers															
Interns															208
Higher (V)	2	2	2			1						2		9	
Higher (F)															
Specialist (V)	1	1	1		1	1	1	1	1	1	1	1	1	13	
Specialist (F)															
Edendale															
Interns	3	3	2			2	1	1	1					13	620
Higher (V)															
Higher (F)	4	4	3			2	1	1	1	1		1		18	
Specialist (V)	3	2	1		1	1		1	1			1		12	
Specialist (F)	1	1	1										1	4	
Edenvale															
Interns	6			1	1			1	1					10	146
Higher (V)												2		2	
Higher (F)	2	2			1							1		6	
Specialist (V)	2	2		1	1			1	1	1	1	1	2	11	
Specialist (F)														2	
False Bay															
Interns	1													1	42
Higher (V)												1		1	
Higher (F)															
Specialist (V)	1	1		1		1	1		1			1		7	
Specialist (F)															
Far East Rand															
Interns	2	2	1			1	1	1						8	474
Higher (V)	3	2	2			1								8	
Higher (F)															
Specialist (V)	1	1	1			1	1	1	1	1	1		1	10	
Specialist (F)												1		1	
Frere															
Interns	2	4	2											8	509
Higher (V)	5	6												11	
Higher (F)	1	1	1			1			1			1		5	
Specialist (V)	3	1	1			1		1	3			2	3	15	
Specialist (F)													1	1	
Germiston															
Interns	1	1	1				1	1		1				6	397
Higher (V)															
Higher (F)	1	1	1	1								1		5	
Specialist (V)	1	1	1	1	1	3	1	1	1	1	1	1	1	15	
Specialist (F)															
Grey (Pietermaritzburg)															
Interns	1	1	1			1		1	1					6	357
Higher (V)	1														
Higher (F)	2	2	1			1		1	1					8	
Specialist (V)	3	3	1		1	1		1	1			2		13	
Higher (F)													1[3]	1	
Krugersdorp															
Interns	1	1	1											3	312
Higher (V)												1		1	
Higher (F)	1	1	1			1	1					1		6	
Specialist (V)	1	1	1		1	1	1	1		1	1			11	
Specialist (F)												1	1	2	

	Medicine	*Surgery*	*Obstetrics*	*Gynaecology*	*Paediatrics*	*Orthopaedics*	*Urology*	*Ophthalmology*	*E.N.T.*	*Dermatology*	*Psychiatry Neurology*	*Anaesthesia*	*Radiology*	*Total*	*Beds*
Livingstone (Port Elizabeth)															474
Interns	2	2	1		1	1								7	
Higher (V)					3									3	
Higher (F)	1	2	1			1				1		1		7	
Specialist (V)	4	5	5		2	2	1	2	3	1	1	2		29	
Specialist (F)													1	1	
National (Bloemfontein)															612
Interns	2	2	1	1		2								8	
Higher (V)															
Higher (F)	1	2	1			1						1		6	
Specialist (V)	3	3	3			2	1	2	2	1	2			19	
Specialist (F)												1	2	3	
Paarl Prov.															200
Interns	1	1				1	1	1	1					6	
Higher (V)												6			
Higher (F)															
Specialist (V)		1				1							2	4	
Specialist (F)															
Port Elizabeth Prov.															304
Interns	1	1	1			1	1	1						6	
Higher (V)															
Higher (F)		1	1			1								3	
Specialist (V)	2	4	3			2	1	2	3	1	1	2	1	22	
Specialist (F)															
Queen Mary (Uitenhage)															134
Interns	1	1	1											3	
Higher (V)	3	3	4											10	
Higher (F)															
Specialist (V)	1	1	1		1	1		1	1	1		1	1	10	
Specialist (F)															
Rondebosch and Mowbray															57
Interns	1	1												2	
Higher (V)															
Higher (F)															
Specialist (V)	2	4	2			2	2	1	1			3		17	
Specialist (F)															
Somerset															253
Interns	2	1	1		1	1			1					7	
Higher (V)															
Higher (F)	2	1	2			1	1					1		8	
Specialist (V)	1	3	5			2	1	1	1	1	1	3		19	
Specialist (F)													2	2	
Vereeniging															154
Interns												3		3	
Higher (V)															
Higher (F)	2	1												4	
Specialist (V)	1	1				1	1		1	1	1			7	
Specialist (F)													1	1	
Victoria															125
Interns	1	1				1	1							4	
Higher (V)															
Higher (F)	1	1				1								4	
Specialist (V)	1	3	2			2	1	2	1	1		2		15	
Specialist (F)															
Voortrekker (Kroonstad)															355
Interns	2	2												4	
Higher (V)			1											1	
Higher (F)		1										1		2	
Specialist (V)	1	2						1						4	
Specialist (F)													1	1	
Woodstock															77
Interns	1	1												2	
Higher (V)															
Higher (F)												1		1	
Specialist (V)	2	2	3			2	1	1	2	1				14	
Specialist (F)													1	1	
Totals															8,984
Interns	59	53	18	3	6	15	8	9	7	2	1	1		182	
Higher (V)	16	14	9		4	2						17		62	
Higher (F)	41	43	26	4	23	15	5	6	4	2		22		206	
Specialist (V)	43	54	34	8	15	37	20	31	33	19	14	31	17	371	
Specialist (F)	13	10	5		3	2	2	1	1	1		12	23	78	
	172	174	92	15	51	71	35	47	45	24	15	83	40	899	8,984

1. Where not otherwise stated gynaecological staff is grouped with the obstetrical staff.

2. At the Conradie Home the chronic sick beds are excluded from the total stated. The staff enumerated are those that attend in the acute wards (but the interns attend also in the chronic wards. In addition to the staff enumerated 10 general practitioners attend at this hospital.

In addition the following staff was returned:

Addington.	*Higher* (*F*):	Out-patients 4, Casualties 2.
	Specialist (*F*):	Thoracic Surgery 1.
	Specialist (*F*):	Physical Medicine 1, Pathology 4.
Baragwanath.	*Higher* (*F*):	Physical Medicine 1, Plastic 1, Thoracic Surgery 1, Neurosurgery 2, Casualties 2.
	Specialist (*V*):	Physical Medicine 1, Plastic 3, Thoracic Surgery 2, Neurosurgery 1.
Boxburg-Benoni.	*Specialist* (*V*):	Physical Medicine 1.
Coronation.	*Specialist* (*V*):	Physical Medicine 1.
Edendale.	*Specialist* (*V*):	Plastic (maxillo-facial) 1.
Krugersdorp.	*Specialist* (*V*):	Physical Medicine 1, Plastic (maxillo-facial) 1.
Livingstone (P.E.).	*Specialist* (*V*):	Thoracic surgery 1.
Vereeniging.	*Higher* (*F*):	Casualty 1.
Victoria.	*Higher* (*F*):	Casualty 1.
Discoverers.	*Specialist* (*V*):	Physical Medicine 1.

TABLE V. GROUP-III HOSPITALS

Hospital	Interns	'Higher'	Visiting Medical Officers	Beds	Obstetrical Beds[1]
Andrew McColm, Pretoria	—	1	—	101	
Barberton	2	—	6	212	13
Barkly West	—	—	2	28	
Beaufort West	2	—	5	66	8
Bernice Samuel, Delmas	—	—	3	20	
Bethal	1	—	6	103	
Bethlehem	1	2	6	139	12
Beulah N. Home, Barkly East	—	—	2	12	2
Bray, Kirstonia (District Vryburg)	—	—	1	22	
Britstown	—	—	2	16	
Burghersdorp	—	—	6	16	4
Butterworth	—	1	6	115	
Cala	—	—	2	32	7
Caledon	—	—	4	65	6
Calvinia	—	—	2	17	
Cathcart Cottage	—	—	3	32	5
Ceres	—	—	8	18	
Citrusdal	—	—	4	20	1
Colesberg	—	—	3	24	4
Cradock Queen's Central	1	—	5	50	
G. J. Crookes, Renishaw	—	3	5	80	
Dordrecht	—	—	4	20	4
Douglas, Wilhelmina N. Home	—	—	2	9	
Duiwelskloof	1	1	2	68	
Elliott Cottage	—	—	2	7	3
Elsie Ballot, Amersfoort	—	—	2	13	
Ermelo	1	1	9[2]	75	
Ficksburg	—	—	5	33	
Fort Beaufort	—	—	3	51	5
George	—	—	6	52	
Grey's, King William's Town	2	1	8	153	
Greytown	—	2	2	64	
Harrismith	2	—	8	62	
Heidelberg	1	1	3[3]	70	
Heilbron	—	—	5	57	
Helpmekaar, Griquatown	—	—	2	18	
Hoopstad (Stephanus Erasmus)	—	—	4	31	5
Hottentots Holland, Somerset West	1	—	6	39	
Humansdorp	1	—	4	50	
Indwe (Private)	—	—	—	20	
Jan en Nellie Keyter	—	—	4	33	
Jansenville (Ex SAWAs Health Memorial)	—	—	3	14	
Keimoes, Carterton Verpleeginrigting	—	—	1	10	3
Kenhardt, Charles N. Home	—	—	2	10	1
Kimberley	6	2	21	349	5
Knysna	—	—	3	56	
Kokstad, East Griqualand (Usher Memorial)	—	—	4	63	
Komgha	—	—	2	9	
Lady Gray N. Home	—	—	1	22	
Ladysmith	4	4	7	407	
Langa, Cape Town	2	1	—	30	
Leydsdorp	—	—	2	21	1
Lichtenburg	—	—	4[4]	108	
Louis Trichardt	—	—	3	28	
Lovedale (Victoria)	3	3	—	120	
Lower Umfolozi, Empangeni	1	4	2	198	
Lydenburg	1	1	3	116	6
Martha Bishop Nursing Home, Marydale	—	—	1	12	
Middelburg, Transvaal	2	1	3	168	14
Molteno	—	—	3	10	4
Montagu	—	—	5	29	
Murraysburg	—	—	—	6	
Newcastle (N R)	1	2	1	126	
Niemeyer Memorial	—	1	—	60	
Nigel, Dunnottar	1	3	5	120	
Odendaalsrus	—	—	3	62	
Olifantshoek Nursing Home	—	—	1	12	
Oudtshoorn (Royal S. Western)	2	—	8	89	12
Ouma Cillie Verpleeginrigting, Kakamas	—	—	2		
Paarl	6[6]	1	20	200[5]	16
Pallotti Nursing Home, George	—	—	—[7]	16	
Pietersburg	2	5	7	212	12
Piet Retief	—	—	4	132	7
Porterville Nursing Home	—	—	—[7]	8	
Port Nolloth	—	—	1	8	
Port Shepstone	—	3	3	143	
Postmasburg	—	—	—	25	
Potchefstroom	1	—	4	142	16
Potgietersrus	—	1	4[8]	66	
Paul Kruger Memorial, Rustenburg	—	1	4	143	2
Prieska	—	—	3	24	
Queenstown Frontier	3	—	9	186	17
Reivilo	—	—	2	26	2
Rita Coetzee Nursing Home, Kirkwood	—	—	4	6	
Riversdale	—	—	5	46	
St. Konrad's, Taungs	1	1	1	150	10
Senekal	—	—	6	46	
Settlers, Grahamstown	2	1	8[9]	—	14
Sir Henry Elliott, Umtata	5	2	7	253	18
Somerset East	—	—	5	49	
Spes Bona, Paulpietersburg	—	—	2	10	
Standerton	2	1	5	109	10
Stanger	—	4	—	115	
Stellenbosch	2	—	9[10]	70	10
Stoffel Coetzee, Smithfield	—	—	3	23	
Stutterheim	—	—	5	30	
Sutherland	—	—	3	15	3
Swartland, Malmesbury	2	—	6[12]	64	
Tayler Bequest, Matatiele	—	—	4	35	
Tayler Bequest, Mount Fletcher	—	1	1	17	
Uniondale	—	—	—	12	2
Upington, Gordonia	—	—	10	45	6
Van der Bijlpark	—	—	—	85	
Ventersdorp	—	—	4	36	8
Vereeniging	—	4	15	283	25
Victoria West	—	1	2	45	6
Virginia	—	—	6	62	
Vrede	—	—	5	30	
Vryheid	—	1	2	119	
Welkom	—	—	6	62	4
Williston (A.C.V.V.)	—	—	2	9	
Witbank	1	1	5[13]	231	
Witzieshoek (Elizabeth Ress)	—	1	1	162[14]	17
Wolmaransstad	2	3	2	98[15]	9
Worcester	2	1	16	64	
Zastron	—	—	4	58	5
Zeerust	—	1	3	68	14

1. Included in previous column.
2. Including 1 orthopaedic surgeon and 1 radiologist.
3. Including 1 anaesthetist.
4. Plus 9 general practitioners.
5. Including 30 paediatric beds.
6. 3 at present.
7. All patients treated by their own practitioners.
8. Including 1 radiologist.
9. Including 1 orthopaedic surgeon.
10. Including 1 orthopaedic surgeon and 1 ophthalmic surgeon.
11. Including 3-4 orthopaedic and 4 gynaecological beds.
12. Including 1 specialist surgeon.
13. Including 1 orthopaedic surgeon and 1 radiologist.
14. Including 24 beds for tuberculosis.
15. Including 4 for orthopaedic cases.

NASIONALE VOEDINGSRAAD

AANBEVOLE MINIMUM DAAGLIKSE DIEETSTANDAARDE*

(Saamgestel deur die Dieetstandaardekomitee van die Raad)

1. In 1935 het die Volkebond[1] die eerste pogings aangewend om die mens se daaglikse behoeftes aan spesifieke voedingstowwe kwantitatief te definieer. Die Voedsel- en Voedingskomitee van die Verenigde State se Nasionale Navorsingsraad het in 1941 'n tabel met baie meer besonderhede gepubliseer, wat gedurende die daaropvolgende jaar deur die Nasionale Voedingsraad by Suid-Afrikaanse toestande aangepas is. Die Amerikaanse syfers is in 1945, 1948 en weer in 1953 hersien. In 1953 het die Nasionale Voedingsraad van Suid-Afrika besluit om sy eie standaarde te hersien en in die onderhawige verslag word die bestaande kennis oor die onderwerp aangepas by die ietwat buitengewone omstandighede van 'n land met 'n gemengde bevolking wat baie verskil in dieetgewoontes en beroep.

Bestaande Standaarde.

2. Die Dieetstandaarde van die Voedsel- en Voedingsraad van die Nasionale Navorsingsraad NRC van die VSA is aanbevelings en nie behoeftes nie, ‚aangesien hulle nie slegs die minimale behoeftes van deursnee persone verteenwoordig nie, maar 'n voedingstofpeil wat gekies is om die individuele afwykings in 'n aansienlike meerderheid van die bevolking te dek . . . Die innames van voedingstowwe wat aanbeveel word, is oor die algemeen hoër as die gemiddelde behoeftes en laer as die hoeveelhede wat nodig mag wees in patologiese toestande of gedurende die rehabilitasie wat op uitputting volg'.[2]

3. Die syfers wat aangegee is deur die Voedingskomitee van die Britse Mediese Vereniging in 1950,[3] is bedoel om voldoende te wees vir die handhawing van 'n goeie voedingsstandaard by gesonde persone. Hierdie standaarde maak ook nie voorsiening vir siekte of herstel nie.

4. Die standaarde van die Kanadese Voedingsraad (1950) is selfs nog laer as die Britse; hulle dien as grondslag vir die bepaling van voedselvoorrade vir groepe, en kan ook gebruik word om die toereikendheid van die voedsel-inname van indiwidue of groepe te beoordeel. ‚Hulle dui ook op 'n voedingsvlak waaronder dit nie aangeneem kan word dat menslike gesondheid instand gehou kan word nie'.[4]

5. In 1950 het 'n komitee van die Voedsel- en Landbouorganisasie (VLO) metodes aanbeveel, waarvolgens kaloriebehoeftes vasgestel kan word. Hulle aanbevelings is bedoel vir toepassing op bevolkings of bevolkingsgroepe. Daar word beklemtoon dat die aanbevelings nie direk van toepassing is op indiwidue nie en selfs wanneer dit op groepe toegepas word, moet plaaslike omstandighede in aanmerking geneem word. Die Komitee het die behoeftes op die ‚fisiologiese peil' oorweeg; hulle aanbevelings verteenwoordig die behoeftes van gesonde indiwidue en is ‚op 'n peil gestel wat 'n aktiewe lewe en 'n hoë mate van produktiwiteit in werksaamhede moontlik maak'. Hulle is gebaseer op 'n ‚kontra'-man (gewig 65 kg., 143 lb.) en -vrou (gewig 55 kg., 121 lb.), ouderdom 25 jaar, wat lewe by 'n gemiddelde jaarlikse uitwendige temperatuur van 10°C (50°F). Wenke word gegee vir die berekening van die behoeftes van indiwidue wat van die ‚kontra-persoon' verskil in liggaamsgrootte, ouderdom, aktiwiteit en omgewingstemperatuur.[5]

Algemene Beginsels.

6. Daar is geen bewyse vir enige rasverskille wat die behoeftes aan voedingstowwe betref nie; gevolglik is daar geen onderskeid tussen die standaarde wat vir blankes en nie-blankes voorgestel word, in die tabel gemaak nie.

7. Die behoeftes aan voedingstowwe van indiwidue word beïnvloed deur geslag, ouderdom, aktiwiteit, ideale gewig en ander faktore. As die werklike gewig as basis geneem word, sal die swaarlywige persoon toeneem in gewig weens die hoër kalorierantsoen waarvoor voorsiening gemaak word, terwyl daar aan die behoeftes van die persoon wat ondergewig is, nie voldoen sal word nie. As die ideale gewig vir lengte, aktiwiteit of liggaamsoppervlakte as maatstaf gebruik kon word, sou die kaloriebehoeftes noukeuriger bepaal kon word; ongelukkig is daar in hierdie opsig 'n gebrek aan voldoende gegewens vir Suid-Afrika.

8. Daar word baie navorsing gedoen oor die kwessie van die noue verwantskap of die wisselwerking tussen die verskillende voedingstowwe. Die inname van een voedingstof beïnvloed moontlik die behoefte aan ander. Op hierdie manier word die tiamienbehoefte bepaal deur die kalorie-inhoud van die dieet; beide folasien en vitamien B_{12} speel 'n rol in die sintese van cholien wat noodsaaklik is vir die instandhouding van lewer-morfologie en -funksie; vitamien D bevorder die absorpsie en metabolisme van kalsium; 'n gebrek aan niasien kom veral voor in diëte wat arm is aan volwaardige proteïen—sodanige proteïen verskaf die essensiële aminosuur, triptofaan, wat 'n voorloper van niasien is.

Die Toepassing en Perke by Gebruik van Dieetstandaarde.

9. Dit is baie belangrik om te onthou dat dieetstandaarde nie as die enigste maatstaf vir die beoordeling van die voedingstoestand van 'n persoon of groep gebruik kan word nie en dat versuim om aan hierdie standaarde te voldoen, nie noodwendig tot gebreksiektes sal lei nie.

10. In hierdie verband is die volgende aanhalings van belang:—

‚Aangesien baie persone wat minder as die aanbevole rantsoen van een of ander voedingstof kry, vir lang periodes gesond kan bly, word dit duidelik dat hierdie rantsoen nie as die enigste maatstaf gebruik moet word om die voedingstoestand van enige bevolking te beoordeel nie'.[6] ‚As hierdie rantsoen gebruik word vir die waardebepaling van diëte, is dit noodsaaklik om te begryp dat hoewel meeste mense wie se inname gelyk is aan, of groter is as die voorgestelde innames, vermoedelik toereikend gevoed is, ly alle persone wat nie soveel inneem nie, nie noodwendig aan wanvoeding nie'.[2]

‚Dit is belangrik dat die standaarde korrek aangewend en hulle beperkings besef word. In die verlede was sommige van die gevolgtrekkings wat gemaak is uit die vergelyking van die peil van voedingsinnames met die dieetstandaarde, ver van juis af. As die resultate van 'n dieetopname toon dat die dieet 'n baie lae kaloriewaarde het, is die gevolgtrekking dat die betrokke groep aan ondervoeding ly, miskien geregverdig. Maar die feit dat die inname van bepaalde voedingstowwe laer is as sekere aanbevole rantsoene, regverdig nie die gevolgtrekking dat 'n deel van enige groep wat ondersoek is, aan wanvoeding ly nie. Onder sulke omstandighede kan die afleiding gemaak word dat daar moontlik wanvoeding bestaan, maar die dieetopname *as sodanig* verskaf geen bewys vir die bestaan daarvan nie.

Die waarde van die inligting is daarin geleë dat dit aandui waar verdere ondersoek nodig mag wees en watter dieetgebreke die meeste aandag moet ontvang in voedsel- en voedingsprogramme'.[7]

Voorgestelde Dieetstandaarde vir Suid-Afrika.

11. Die standaarde wat hier voorgestel word, behoort as voldoende vir die onderhoud van gesondheid beskou te word, sonder dat dit voorsiening maak vir 'n veiligheidsgrens vir swak gesondheid of vir groot indiwiduele verskille in absorpsie en metabolisme. Die fisiese veranderinge wat die normale liggaam met ouderdom en aktiwiteit ondergaan, is in aanmerking geneem; byvoorbeeld, vanaf die ouderdom van 10 jaar word die behoeftes van seuns en dogters apart oorweeg, weens verskille in gewig, aktiwiteit en veranderings wat vroeër by dogters as by seuns intree.

12. Die syfers wat aangegee word, is vir voedingstowwe in voedsels soos hulle ingeneem word, en maak nie voorsiening vir verliese gedurende vervoer, opberging, bereiding en bediening nie. Min is bekend aangaande die mate van hierdie verliese. Dit is waarskynlik dat daar gewoonlik min of geen verlies aan proteïen of koolhidraat is nie, maar wanneer sekere gaarmaakmetodes gebruik word, bv. rooster en in die oond braai, kan daar 'n groot verlies aan vet wees. Mineraalsoute, soos kalsium kan uit groente uitgeloog word wanneer dit in water gekook word. Die verliese aan vitamiene kan baie hoog wees, veral dié aan askorbiensuur. Syfers verkry uit lektuur toon die volgende gemiddelde verliese in groentes gedurende die gaarmaak, na gelang van die metode wat gebruik word.[8]

Karoteen	5—21%
Tiamien	9—37%
Ribofiavien	12—37%
Niasien	9—41%
Askorbiensuur	27—47%

* Het in Engels verskyn in verlede week se Tydskrif (S. Afr. T. Geneesk. 1956, **30**, 108).

Die lae syfer is van toepassing op die stoommetode (waterless) en die hoë syfer wanneer die voedselsoort onder water gekook word. Verliese is te wyte aan uitloging (riboflavien en niasien), werklike vernietiging (askorbiensuur) of albei (tiamien).

Askorbiensuur word in 'n toenemende mate vernietig deur 'n oksiderende ensiem wat vrygestel word in baie vrugte en groente nadat hulle gepluk is. Dit is dus baie wenslik dat die tyd tussen die pluk, bereiding en bediening van hierdie produkte so kort as moontlik moet wees.

13. Die gewig van die gemiddelde Suid-Afrikaanse man of vrou is nie bekend nie, maar daar is besluit om die arbitrêre syfer van 160 lb. (73 kg.) vir die kontraman van 25 jaar en 130 lb. (59 kg.) vir die kontravrou van dieselfde ouderdom, aan te neem.

In Tabel II word aanpassings van kaloriebehoeftes vir gewigte wat verskil van dié van die kontraman en -vrou, aangegee.

14. In die hoër ouderdomsgroepe, veral as die werk van 'n sittende aard is, kan die kalorieë afkomstig van beide koolhidrate en vette met voordeel verminder word om swaarlywigheid en sekere degeneratiewe siektes te voorkom, maar die inname van ander voedingstowwe moet nie verminder word nie. Daarenteen kan dit wel wees dat die inname van ander voedingstowwe as koolhidrate en vette eerder vermeerder as verminder behoort te word, omdat die liggaam van 'n anaboliese in 'n kataboliese toestand verander.

15. Dit is goed bekend dat die behoeftes gedurende swangerskap en laktasie groter is as dié van nie-swanger en nie-sogende vrouens. In sekere gevalle (bv. kalorieë, proteïen en kalsium) is dit moontlik om die bykomende behoeftes te bereken volgens die bekende samestelling van die produkte van bevrugting of van moedersmelk. In die geval van baie ander voedingstowwe is weinig bekend aangaande die vereiste hoeveelhede, hoewel dit algemeen aangeneem word dat hulle vermeerder moet word.

Kalorieë.

16. Omdat uitwendige temperature die kaloriebehoefte beïnvloed, moet groot afwykings in aanmerking geneem word. Om hierdie standaarde op te stel is 'n gemiddelde temperatuur van 60°F geneem en die metode van aanpassings wat deur die VLO[5] vir uitwendige temperature gebruik word, is gevolg.

17. Op eenjarige leeftyd is die behoefte naasteby 45 kalorieë per pond liggaamsgewig (100 kalorieë/kg.). Bo hierdie leeftyd verminder die groeisnelheid en daal die behoefte per eenheid liggaamsgewig dienooreenkomstig.

Onder 10 jaar word die kaloriebehoeftes van seuns en dogters as dieselfde beskou. Bokant hierdie ouderdom word hulle behoeftes op verskillende peile vasgestel. Gedurende die tydperk van puberteit en adolessensie is 'n verhoogde eetlus dikwels baie opvallend. Mits die fisiese aktiwiteit groot is, moet die verhoogde eetlus bevredig word deur vergrote innames van kaloriegewende en ander voedingstowwe tot op 'n vlak wat dikwels gelyk is aan dié wat nodig is vir volwassenes wat swaar arbeid verrig.

Arbeiders in hierdie land het betreklik meer kalorieë nodig as die Amerikaanse arbeider omdat in laasgenoemde land baie meer gebruik gemaak word van werkbesparende toestelle.

18. Die mening neem toe dat gewigstoename nadat volwassenheid bereik is, (25 jaar) nie fisiologies is nie en vermy moet word. Die toename in gewig op middeljarige leeftyd is grootliks toe te skryf aan 'n vermeerdering van die vet-inhoud van die liggaam en gaan nie gepaard met 'n toename in aktiewe protoplasmiese massa nie, maar selfs met 'n verlies daarvan. Die rede vir 'n vermindering van die aktiewe protoplasmiese massa op middeljarige leeftyd kan gedeeltelik van endokriene-oorsprong wees, maar kan byna sekerlik gedeeltelik toegeskryf word aan verminderende aktiwiteit; dus behoort die kalorie-inname verminder te word met toenemende ouderdom. Die aanbeveling van die Voedsel- en Voedingsraad van die VSA van 'n vermindering van 5% vir elke dekade word aangeneem.[2] Op hierdie grondslag is die gemiddelde behoefte op 35, 45, 55, en 65 jaar, onderskeidelik 95%, 90%, 85% en 80% van die kaloriebehoefte op 25 jaar.

Proteïen.

19. Die standpunt dat 'n persoon minder proteïen nodig het as wat voorheen gemeen is, wen vinnig veld.[9,10] Dit is egter belangrik dat wanneer die dieet oorwegend uit graansoorte, soos koring en mielies, bestaan, dit aangevul moet word óf met dierlike voedselsoorte óf met peulgewasse, en wanneer mielies hoofsaaklik gebruik word, moet die proteïenstandaard effens verhoog word. Met die oog op die belangrikheid van proteïen vir die bou en funksionering van die liggaam en vir beskerming teen tuberkulose en moontlik ander infeksies, is dit raadsaam dat die standaard vir die volwasse man nie laer moet wees as 65 g. nie, waarvan minstens 1/3de van dierlike oorsprong moet wees, wat gedeeltelik vervang kan word deur proteïen uit droë peulgewasse. Dean[11] meen dat 'n graanpeulgewasmengsel in 'n groot mate die melkproteïen in die dieet van kinders kan vervang.

As daar nie in die kaloriebehoeftes voorsien word nie, word 'n deel van die proteïen gemetaboliseer om die tekort aan te vul en derhalwe is daar minder beskikbaar vir die opbouing, beskerming en regulering van die liggaam.

20. Die proteïenbehoefte gedurende die 3de trimester van swangerskap en gedurende borsvoeding is aansienlik hoër en omdat dit noodsaaklik is om volwaardige proteïen te vervaardig, word die rantsoen van dierlike proteïen vermeerder tot 40% van die totaal.

21. By die kind is die behoefte per eenheid liggaamsgewig groter as by die volwassene en die kwaliteit van die proteïen is van nog groter belang. Gedurende die afgelope dekade was dit gebruiklik om, in terme van koeimelkproteïen, vir kinders 3·5 g. proteïen per kg. of 1·6 g. per lb. liggaamsgewig aan te beveel.

Tydens die onlangse Konferensie oor Proteïenbehoeftes (Josiah Macy, Jnr. Foundation met VLO en WGO van VVO, 19-24 Junie 1955, Princeton, New Jersey, VSA) is die mening uitgespreek dat hierdie syfers onnodig hoog mag wees. Die gevolgtrekkings waartoe geraak is en meer spesifiek, die feite waarop dit berus, is nie tot die beskikking van die Komitee nie. Die voorgestelde laer standaarde word ook net in terme van koeimelkproteïen aangegee en geen ooreenkomstige syfers vir ander dierlike- of planteproteïen is beskikbaar nie. Die Komitee het dus by die vroeëre standaarde gehou.

Kalsium.

22. Daar is toenemende bewyse dat die menslike liggaam homself aanpas om ekonomies en doeltreffend te bestaan op 'n peil van kalsiuminname wat aansienlik laer is as die rantsoen wat gewoonlik aanbeveel word.[3] Onder die inboorlingbevolkings van Suid-Afrika is die innames dikwels 'n derde van sulke rantsoene sonder enige merkbare nadelige gevolge. Ongelukkig is daar geen metode om die peil van kalsiumopberging in die liggaam te bepaal nie. Voorts skyn daar, in sterk teenstelling met ander voedingstowwe waarvan die inname hier aanbeveel word, geen bewys te wees van enige sindroom wat uitsluitlik deur 'n kalsiumgebrek veroorsaak word of net deur kalsium-byvoeging geneesbaar is nie. Om 'n rantsoen van 'n dieetbestanddeel aan te beveel om beskerming te bied teen ongespesifiseerde stigmata skyn 'n twyfelagtige handelwyse te wees. Metaboliese studies op goedgevoede blankes, verskaf egter bewyse dat daar 'n neiging tot 'n negatiewe kalsiumbalans is as die inname kleiner as 10 mg. kalsium per kg. liggaamsgewig per dag is. In die lig van ons huidige kennis is daar dus besluit om hierdie peil van inname aan te beveel vir die volwasse man en die nie-swanger en nie-sogende vrou.[12]

Die aanbevole kalsiumrantsoen vir swangerskap en laktasie word bereken volgens die hoeveelhede wat nodig is om die fetus daarvan te voorsien en om die kalsiumpeil in die moedersmelk te handhaaf. Die standaarde vir kinders is bereken volgens die toename in kalsium in die liggaam gedurende groei soos opgegee in die lektuur.

Yster.

23. Die presiese behoeftes aan yster is nog nie behoorlik vasgestel nie, maar dit is bekend dat 'n ystergebrek of hipochromiese anemie meer by vrouens as by mans voorkom. As die inname van ander voedingstowwe bevredigend is, is daar geen moeilikheid om in die aanbevole rantsoen van yster te voorsien nie. Daar is bewys dat 3 mg. meer per dag gedurende swangerskap nodig is. Dit blyk dat 'n groot persentasie Bantoes teoreties 'n oormaat yster, wat afkomstig is van ysterpotte wat gebruik word, inneem.

Vitamien A.

24. Vitamien A kan in die dieet in die vorm van sy voorloper, karoteen, of as aktiewe vitamien A teenwoordig wees. Daar sal dus na die voedingstof òf as karoteen òf as vitamien A verwys word. Een I.E. vitamien A is gelyk aan 0·3 mikrogram vitamien *A-alkohol* of 0·6 mikrogram *β*-karoteen. Weens die onsekerheid omtrent die mate van omsetting van karoteen in vitamien A, is dit wenslik dat 'n deel, miskien soveel as 1/3de daarvan, as vitamien

TABEL I. AANBEVOLE MINIMUM DAAGLIKSE DIEETSTANDAARDE

	Kalorieë (*a*)	*Proteïen* g.	*Kalsium* g.	*Yster* mg.	*Vit. A* I.E.	*Tiamien* mg.	*Riboflavien* (*o*) mg.	*Niasien* (*q*) mg.	*Askorbiensuur* mg.
Man (gemiddelde gewig 160 lb.)									
Matig aktief ..	3,000	65	0·7	9	4,000 (*k*)	1·0	1·6	15	40
Sittende werk ..	2,300	65	0·7	9	4,000 (*k*)	0·8	1·6	12	40
Swaar arbeid ..	4,500	65	0·7	9	4,000 (*k*)	1·6	1·6	18	40
Vrou (gemiddelde gewig 130 lb.)									
Matig aktief ..	2,300	55	0·6	12	4,000 (*k*)	0·8	1·4	12	40
Sittende werk ..	2,000	55	0·6	12	4,000 (*k*)	0·7	1·4	11	40
Swaar arbeid ..	2,800 (*b*)	55	0·6	12	4,000 (*k*)	1·0 (*b*)	1·4	15 (*b*)	40
Swangerskap (laaste trimester)	(*c*)								
Matig aktief ..	2,600	80 (*g*)	1·5	15	5,000 (*l*)	0·9	2·0	14	55
Sittende werk ..	2,200	80 (*g*)	1·5	15	5,000 (*l*)	0·9	2·0	13	55
Swaar arbeid ..	3,200	80 (*g*)	1·5	15	5,000 (*l*)	1·1	2·0	15	55
Laktasie	(*d*)	80 (*g*)	(*i*)	15	6,000 (*l*)	(*n*)	(*p*)	15	55
Kinders									
0– 3 maande ..	55 cal/lb.	1·6 g/lb. (*h*)	0·8 (*j*)	6 (*h*)	1,500 (*m*)	0·2	0·5 (*h*)	2 (*r*)	20 (*s*)
4– 9 maande ..	50 cal/lb.	1·6 g/lb. (*h*)	0·8 (*j*)	6 (*h*)	1,500 (*m*)	0·2	0·8 (*h*)	4	25 (*s*)
10–12 maande ..	45 cal/lb. (*e*)	1·6 g/lb. (*h*)	0·8 (*j*)	6 (*h*)	1,500 (*m*)	0·35	0·9 (*h*)	4	30 (*s*)
1– 3 jaar.. ..	1,100	40	0·6	7	2,000 (*l*)	0·4	1·0	6	40
4– 6 jaar.. ..	1,500	45	0·7	8	2,500 (*l*)	0·5	1·1	8	40
7–10 jaar.. ..	1,900	55	0·8	10	3,000 (*l*)	0·7	1·4	10	40
Dogters									
10–12 jaar.. ..	2,400 (*f*)	70	1·0	12	3,000 (*l*)	1·0	1·8	12	40
13–15 jaar.. ..	2,600	75	1·2	15	4,000 (*l*)	1·1	1·9	15	40
16–20 jaar.. ..	2,400 (*b*)	70	1·2	15	4,000 (*l*)	1·0 (*b*)	1·8	15 (*b*)	40
Seuns									
10–12 jaar.. ..	2,400	65	0·8	12	3,000 (*l*)	1·0	1·6	12	40
13–15 jaar.. ..	3,000	75	1·3	15	4,000 (*l*)	1·2	1·9	15	40
16–20 jaar.. ..	3,700	90	1·3	15	4,000 (*l*)	1·5	2·3	15	40

(*a*) Hoewel 'n enkel syfer in elke geval aangegee word, moet dit as 'n gemiddelde beskou word, waarvan daar indiwidueel afgewyk kan word. Die kalorierantsoen moet in elk geval by die behoeftes van die indiwidu aangepas word, sodat sy ideale gewig bereik en gehandhaaf kan word. (Sien Tabel 2.)

(*b*) Dit mag wees dat hierdie waarde aansienlik vermeerder moet word in die geval van vrouens wat swaar arbeid verrig, byvoorbeeld dié wat op die lande werk.

(*c*) Die tempo van die moeder se basaalmetabolisme word nie deur swangerskap verander nie en die vermeerderde metaboliese behoeftes is toe te skryf aan die fetus en die vermeerderde gewig van die moeder.

(*d*) Voeg 120 kalorieë by vir elke 100 ml. melk wat geproduseer word, bv. op een maand is die gemiddelde produksie van melk ongeveer 700 ml. en voersiening behoort gemaak te word vir 840 kalorieë. Op 4 maande is die produksie omtrent 1 liter en behoort daar voorsiening gemaak te word vir 1,200 kalorieë.

(*e*) Maak voorsiening vir ongeveer 1,000 kalorieë vir groepe onder een jaar.

(*f*) Voorsiening is gemaak vir minder aktiwiteit in vergelyking met seuns, maar die groter behoefte van vroeër puberteitsveranderings weeg hierteen op.

(*g*) Voorsiening word gemaak vir 'n bykomende rantsoen van 25 vir swangerskap en waar 'n baba geheel en al deur die moeder gevoed word. Die helfte van die rantsoen behoort van dierlike oorsprong te wees.

(*h*) Die aanbevelings vir suigelinge het betrekking op proteïen, yster, ens., hoofsaaklik uit koeimelk of kommersiële melkpreparate verkry.

(*i*) Voeg 120 mg. kalsium by vir elke 100 ml. melk geproduseer; op die ouderdom van 4 maande byvoorbeeld sal die rantsoen 1·2 gram wees.

(*j*) Waar moedersmelk nie gebruik word nie.

(*k*) Uitgaande van die veronderstelling dat 1/3de teenwoordig is as vitamien A; of 5,000 I.E., as 1/5de teenwoordig is as vitamien A.

(*l*) Verkieslik 1/3de as vitamien A in elke geval.

(*m*) Hoewel dit baie varieer, is daar bewyse dat die vitamien A-inname deur moedersmelk omtrent 2,000 I.E. op 2 maande en 2,500 I.E. op 4 maande is, maar daar is nog nie vasgestel of hierdie hoë nnames noodsaaklik is nie.

(*n*) Maak voorsiening vir 0·4 mg. vir elke bykomende 1,000 kalorieë (sien (*d*)).

(*o*) Sien paragraaf 26.

(*p*) Waar 'n baba geheel en al deur die moeder gevoed word, behoort die rantsoen riboflavien 2·0 mg. te wees.

(*q*) Sien paragraaf 27.

(*r*) Hierdie syfer is gebaseer op die bekende niasien konsentrasie in moedersmelk en met in agneming van die feit dat pellagra onbekend is onder kinders wat op moedersmelk gevoed word.

(*s*) Gebaseer op die vitamien C-konsentrasie van moedersmelk.

A teenwoordig moet wees. As dit nie moontlik is nie, is 'n ruimer toelaag van karoteen wenslik.

Tiamien.

25. Die algemene mening skyn te wees dat volwassenes 'n minimum van 0·23 mg. tiamien per 1,000 kalorieë nodig het. Hierdie standaard word maklik bereik wanneer diëte ongesifte of verrykte graanvoedsels of 'n oorvloed goeie kwaliteit proteïen bevat. Daar is gevind dat 0·35 mg./1,000 kalorieë voldoende is om toe te laat vir opberging in die weefsels.[13] Weens die groter eise van swangerskap en laktasie moet die rantsoen gedurende hierdie periode effens hoër as 0·35 mg./1,000 kalorieë wees. Die tiamienrantsoen vir kinders is ook op kalorie-inname gebaseer. Gedurende puberteit en adolessensie word 'n ekstra rantsoen gegee om te voorsien in die hoër fisiologiese behoeftes.

Riboflavien.

26. Hoewel nog nie vasgestel is dat die behoefte aan riboflavien direk met die proteïenbehoefte verband hou nie, is daar besluit om die Nasionale Navorsingsraad se aanbeveling[2] van 0·025 mg. riboflavien per g. proteïen aan te neem.

Niasien.

27. Daar is 'n intieme verwantskap tussen die metaboliese bane van triptofaan en niasien. Hoewel algemeen aangeneem word dat die niasienbehoefte omtrent 10 keer soveel as dié aan tiamien is, is hierdie rantsoen verhoog vanweë die feit dat die dieet van die meerderheid van die inwoners van hierdie land gewoonlik arm aan triptofaan is.

Askorbiensuur.

28. Van al die vitamiene is askorbiensuur die gevoeligste vir oksidasie en hitte; 'n groot persentasie kan vernietig word met die vervoer, opberging en bereiding van groente en vrugte.

Daar is 'n noue verwantskap tussen askorbiensuur en ander voedingstowwe, bv. dié verantwoordelik vir die vorming en instandhouding van weefsel en vir die ontwikkeling van die bloedselle. Voorts kan die toevoer van askorbiensuur wissel met die seisoene in bepaalde dele van die land. Die aanbevole rantsoene is dus op 'n peil heelwat hoër as die Kanadese syfers gestel, maar aansienlik laer as dié van die Nasionale Navorsingsraad[2] van die VSA, wat as onnodig hoog beskou word.[14]

Vitamien D.

29. Vitamien D is noodsaaklik vir die absorpsie en metabolisme van kalsium en fosfor. As die moontlikheid bestaan dat die blootstelling aan sonlig onvoldoende is, of dat die sonlig minder aktief is (soos in die Westelike Provinsie gedurende die winter) kan 'n terapeutiese inname van 400 I.E. per dag vir suigelinge en swanger en sogende vroue voorgeskryf word. Nog 'n rede vir vitamien D-terapie is die grootskaalse gebruik van onverfynde grane wat fitiensuur bevat wat die absorpsie van kalsium mag benadeel.

Oorweging van Voedingstowwe wat nie getabelleer is nie.

30. *Vet.* Daar word aan die hand gedoen dat die peil van vet in die dieet 20-30% van die totale kalorie-inname moet wees, en vir kinders, adolessente en moontlik vir baie aktiewe volwassenes kan dit 30-40% van die totale kalorieë uitmaak. Onder die ekonomies bevoorregtes is daar die neiging tot onwenslik hoë innames van vet wat, na beweer word, potensieel gevaarlik is, tenminste op middeljarige leeftyd in die geval van persone wat sittende werk verrig. Volgens sekere gesaghebbendes is die hoogste wenslike vet-inname onder hierdie omstandighede vermoedelik omtrent 30% van die totale kalorieë. In die moontlike behoefte aan essensiële vetsure (linoleïen-, linoleen- en arachidoniese suur) word daar maklik voorsien as vet 20% of meer van die kalorieë verskaf.

31. *Koolhidrate.* Onder die Westerse bevolkings verskaf koolhidrate gewoonlik minder as die helfte van die energiewaarde van die dieet. Onder die armer groepe van sulke bevolkings egter, sowel as in die algemeen onder bevolkings wat in tropiese en semi-tropiese gebiede woon, kan koolhidrate 3/4 of meer van die kalorieë bydra. Onder sulke omstandighede moet die huidige gebruik om graansoorte (die hoofbron van koolhidrate) te verfyn, om meel met 'n verbeterde voorkoms, smaaklikheid en houvermoë te produseer, betreur word. Nie alleen word die kwaliteit van die proteïen verminder nie, maar daar is ook verliese aan sekere mineraalsoute en vitamiene; veral tiamien raak verlore en hierdie voedingstof, in moontlike kombinasie met ander voedingstowwe, is noodsaaklik vir koolhidraatmetabolisme.

32. *Piridoksien en ander lede van die vitamien-B kompleks.* Geen rantsoene vir hierdie vitamiene word opgegee nie, omdat aangeneem word dat daar geen gebrek aan ander lede van hierdie groep behoort te wees nie as daar toereikende voorsiening vir proteïen en die reedsgenoemde B-vitamiene uit natuurlike bronne gemaak word.

TABEL II. VERWANTSKAP TUSSEN KALORIEBEHOEFTES EN WENSLIKE GEWIG:[5]

Gewig lb.	*Man*		*Vrou*	
	Kalorieë	*Persentasie van die kwota vir die kontraman*	*Kalorieë*	*Persentasie van die kwota vir die kontravrou*
240 ..	4,035	135		
230 ..	3,912	130		
220 ..	3,787	126		
210 ..	3,660	122		
200 ..	3,532	118	3,151	137
190 ..	3,402	113	3,034	132
180 ..	3,271	109	2,918	127
170 ..	3,137	105	2,798	122
160 ..	**3,000**	**100**	2,677	116
150 ..	2,863	95	2,554	111
140 ..	2,723	91	2,423	105
130 ..	2,579	86	**2,300**	**100**
120 ..	2,433	81	2,170	94
110 ..	2,283	76	2,036	88
100 ..	2,125	71	1,900	83
90 ..	1,972	66	1,759	77
80 ..	1,809	60	1,614	70

Kontraman: 160 lb **Kontravrou: 130 lb**

Die Dieetstandaarde-Komitee van die Nasionale Voedingsraad: F. W. Fox (Voorsitter), J. F. Brock, J. T. Irving, J. M. Latsky, W. A. Odendaal, A. J. du Plessis, A. R. P. Walker, L. J. Louw (Mej.).

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THE SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL

RULES REGARDING SPECIALISTS

RULES REGARDING THE REGISTRATION OF THE SPECIALITIES OF MEDICAL PRACTITIONERS AND DENTISTS, THE REQUIREMENTS TO BE SATISFIED BEFORE THEIR SPECIALITIES CAN BE REGISTERED, THE CONDITIONS WHICH SHALL EXEMPT ANY PERSON FROM SUCH REQUIREMENTS AND THE CONDITIONS GOVERNING THE PRACTICE OF MEDICAL PRACTITIONERS AND DENTISTS WHOSE SPECIALITIES HAVE BEEN REGISTERED (GOVERNMENT NOTICE NO. 129 OF 27 JANUARY 1956).

The Minister of Health, in exercise of the powers conferred on him by sub-section (4) of section *ninety-four* of the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928), as amended, has approved of the following rules made by the South African Medical and Dental Council under paragraph (*r*) of sub-section (2) of the said section (Government Notice No. 129 of 27 January 1956).

1. In these rules 'the Act' means the Medical, Dental and Pharmacy Act, 1928 (Act No. 13 of 1928), as amended, and any expression to which a meaning has been ascribed in the Act shall, when used in these rules, bear the same meaning.

2. In these rules, unless inconsistent with the context, 'speciality' means a particular branch of medicine or dentistry; and 'specialist' means a medical practitioner or dentist whose speciality or specialities have been registered under section *thirty-three* of the Act and who confines his practice to such speciality or specialities.

3. The following specialities shall be registrable in terms of section *thirty-three* of the Act:

(*a*) MEDICAL PRACTITIONERS

Speciality	*Designation*
Surgery	Specialist Surgeon.
Medicine	Specialist Physician.
Obstetrics and Gynaecology or Midwifery and Diseases of Women	Specialist Obstetrician and Gynaecologist or Specialist in Midwifery and Diseases of Women.
Anaesthetics	Specialist Anaesthetist.
Dermatology or Diseases of the Skin	Specialist Dermatologist or Specialist in Diseases of the Skin.
Neurology or Diseases of the Nervous System	Specialist Neurologist or Specialist in Diseases of the Nervous System.
Neuro-Surgery or Surgery of the Nervous System	Specialist Neuro-Surgeon or Specialist in Neuro-Surgery.
Ophthalmology or Diseases of the Eye	Specialist Ophthalmologist or Specialist in Diseases of the Eye.
Orthopaedics	Specialist Orthopaedist.
Otorhinolaryngology or Diseases of the Ear, Nose and Throat	Spesialist Otorhinolaryngologist or Specialist in Diseases of the Ear, Nose and Throat.
Pathology	Specialist Pathologist.
Pediatrics or Diseases of Children	Specialist Pediatrician or Specialist in Diseases of Children.
Physical Medicine	Specialist in Physical Medicine.
Plastic and Maxillo-facial Surgery	Specialist Plastic and Maxillo-facial Surgeon.
Psychiatry or Mental Disorders	Specialist Psychiatrist or Specialist in Mental Disorders.
Radiology	Specialist Radiologist.
Diagnostic Radiology	Specialist Diagnostic Radiologist.
Therapeutic Radiology	Specialist Therapeutic Radiologist.
Thoracic Surgery	Specialist Thoracic Surgeon.
Urology or Diseases of the Genito-urinary System	Specialist Urologist or Specialist in Genito-urinary Diseases.
Venereology or Venereal Diseases	Specialist Venereologist or Specialist in Venereal Diseases.

(*b*) DENTISTS

Speciality	*Designation*
Orthodontia	Specialist Orthodontist.
Maxillo-facial and Oral Surgery	Specialist Maxillo-facial and Oral Surgeon.

4. A medical practitioner or dentist may not have more than one speciality registered against his name or practise more than one speciality simultaneously except in the following cases in which the specialities bracketed together shall be regarded as associated specialities and a medical practitioner or dentist (on compliance with the conditions hereinafter laid down) may have one or both such specialities registered and may practise both:

Speciality	*Designation*
Dermatology or Diseases of the Skin	Specialist Dermatologist or Specialist in Diseases of the Skin.
Venereology or Venereal Diseases	Specialist Venerologist or Specialist in Venereal Diseases.
Neurology or Diseases of the Nervous System	Specialist Neurologist or Specialist in Diseases of the Nervous System.
Psychiatry or Mental Disorders	Specialist Psychiatrist or Specialist in Mental Disorders.
Diagnostic Radiology	Specialist Diagnostic Radiologist.
Therapeutic Radiology	Specialist Therapeutic Radiologist.

5. A medical practitioner who desires to have his speciality entered in the register shall be required—

(*a*) to hold a higher qualification in the form of a degree or diploma related to the speciality concerned; such degree or diploma to be of a standard acceptable to the Council;

(*b*) to submit proof to the Council that a period of at least six years has elapsed after obtaining a qualification which entitled him to registration as a medical practitioner or a 'residental medical officer' in terms of the regulations framed under the provisions of section *twenty-two* of the Act or as an intern in terms of the regulations framed under the provisions of section *twenty-five* of the Act;

NOTE.—The year which a practitioner served as a 'resident medical officer' or an intern may be one of the six years referred to above.

(*c*) to submit proof to the Council that, subsequent to having registered as a medical practitioner, he has spent either, (i) at least two years in general practice, or (ii) in lieu thereof has obtained such other experience as the Council may from time to time; determine work performed during the first year after qualification will not count towards experience in lieu of general practice.

NOTE (1).—It is desirable that this experience be obtained before the clinical experience in the relevant speciality prescribed in paragraph (*d*) hereunder.

NOTE (2).—In the case of persons undertaking training in lieu of general practice in terms of paragraph (*c*) above, at least one year's experience must be obtained in general medicine and/or general surgery.

(*d*) to satisfy the following additional specific requirements in the speciality which he wishes to have registered against his name:

(i) In the specialities medicine, surgery; obstetrics and gynaecology; anaesthetics; dermatology; neurology; neuro-surgery; ophthalmology; orthopaedics; otorhinolaryngology; pediatrics; physical medicine; plastic and maxillo-facial surgery; psychiatry; radiology; diagnostic radiology; therapeutic radiology; thoracic surgery; urology; venereology—that he has had three years satisfactory clinical experience as the holder of a clinical appointment under the control of the department in a teaching hospital;

(ii) in the speciality pathology—that he has had three years' satisfactory experience in a teaching institution or university recognized by the Council in all the subjects of general pathology.

NOTE (1).—Experience at a hospital or institution of less than three months' duration will not be regarded as satisfactory experience as prescribed in the rules.

NOTE (2).—If a practitioner has had two years' satisfactory experience in his speciality in an approved hospital or institution, he may be given a maximum credit of twelve months' specialistic training (or if less than two years' experience, not not less than six months' experience, a proportionate exemption, provided that the total exemption does not exceed twelve months).

NOTE (3).—Credit may be given for general practice depending on the quality and type of general practice done, provided such practice was done for a period of at least eight years. (This note is not applicable to the speciality pathology.)

NOTE (4).—Where a medical practitioner wishes to specialize in both dermatology and venereology the periods specified under the rule prescribing clinical specialistic experience in dermatology and venereology each become two years making a total of four years; provided that the total period spent in a teaching hospital shall not be less than three years.

NOTE (5).—Where a medical practitioner wishes to specialize in both neurology and psychiatry, the total period of clinical specialistic training becomes five years with a minimum of two years' experience in each speciality.

NOTE (6).—In the speciality psychiatry, clinical experience in terms of the above rules shall include a minimum of twelve months' experience in a mental hospital of which at least six months must be in a mental hospital which is also a teaching hospital; in the case of work done in a mental hospital which is not also a teaching hospital, the provisions of Note (2) above, will apply.

NOTE (7).—Where a practitioner wishes to specialize in both diagnostic radiology and therapeutic radiology the total period of clinical specialistic training becomes five years, with a minimum of two years' experience in each speciality.

6. Notwithstanding anything to the contrary in these rules contained, it shall be lawful for the Council to register the speciality of a medical practitioner who has not fully complied with the requirements of these rules, if the Council, after due enquiry, is satisfied that such medical practitioner is competent to practise as a specialist.

7. A dentist who desires to have his speciality inserted in the register, and who was not practising such speciality prior to the promulgation of these rules, shall be required to hold a degree or diploma indicating to the satisfaction of the Council a standard of professional education related to the speciality concerned higher than that prescribed for registration as a dentist, and to submit documentary proof to the Council as follows:

(*a*) That he has held a registrable qualification for a period of at least five years; and

(*b*) that he has spent at least two of these years in general practice, or in lieu thereof has obtained such other experience as the Council may from time to time determine;

(*c*) that he has spent either two years full-time, or longer part-time period covering the same prescribed course, in a recognized university, dental school, hospital, or similar institution or department, which provides satisfactory opportunity for the study of the particular speciality.

8. A medical practitioner or dentist may, at his own written request, have the name of his speciality removed from the register of medical practitioners or dentists, as the case may be.

9. A medical practitioner or dentist whose speciality has been registered by the Council shall confine his practice entirely to the speciality or associated specialities registered against his name and the retention of that speciality or those specialities in the register against his name shall be contingent on his doing so: provided, however, that it shall be incumbent on a specialist to perform without extra charge, such other examinations as are usually performed by general practitioners, and provided further that a medical practitioner or dentist whose speciality has been registered shall not charge for examinations or procedures which properly fall under other specialities.

10. A specialist receiving a patient sent to him by another practitioner shall behave as a consultant and send the patient back to such practitioner unless specially asked by such practitioner to continue to treat the case.

11. A specialist may treat any person who may come to him direct for consultation.

12. Rules 8, 9, 10, 11 and 12 of the rules for the forms to be filled in and the documents to be submitted by applicants for registration or for restoration to the registers and for forms of certificate issuable under the Act, published in Government Notice No. 2198 of 1930, as amended, are hereby rescinded.

SOUTH AFRICAN MEDICAL AND DENTAL COUNCIL

REGISTRATION OF MEDICAL AND DENTAL STUDENTS AND OF INTERNS

It is announced in Government Notice No. 128 of 27 January 1956 that his Excellency the Governor-General has been pleased after considering a recommendation by the South African Medical and Dental Council to amend the regulations regarding the registration of medical students, published under Government Notice No. 1083 of 1942, as amended, as follows:

(*a*) By the deletion of the first paragraph of Note C in Chapter I, which read as follows: 'The Council will recognize ordinary certificates of conditional exemption granted by the Joint Matriculation Board only if the subject to be passed in order to obtain the full exemption certificate is one of botany, chemistry, physics or zoology'.

(*b*) By the substitution in Regulation 1 of Chapter IV of the words 'twenty-one' for the words 'twenty-two'. Regulation 1 read as follows: 'No person shall be eligible for registration as an intern or a medical practitioner .... unless he has attained the age of twenty-two years'.

THE PRINCIPLES, METHODS AND MEDIA OF PUBLIC HEALTH EDUCATION

A RESIDENTIAL SEMINAR IN LONDON

The Secretary for Education, Arts and Science, Pretoria, has circulated the following information supplied by the Central Council for Health Education, London.

A Residential Seminar on the Principles, Methods and Media of Health Education will be held for Public Health Workers from overseas, at Passfield Hall, Endsleigh Place, London, W.C. 1, on 17-21 April 1956, arranged by the Central Council for Health Education.

Programme. The programme has been specially designed to help health workers to make the best possible use of all health education opportunities, to improve their knowledge and skills in the use of educational methods and media, and to provide guidance in the preparation of community health education programmes.

Delegates. The Seminar is intended for professional health personnel, educationists, and auxiliary workers concerned with health education of the public. It has been planned primarily for the benefit of delegates from abroad who will be coming to England to participate in the Health Congress of the Royal Society of Health, and for this reason will be held during the week immediately preceding the Congress so that delegates who wish to do so can attend both meetings with a minimum of expense and travelling.

Accommodation. Delegates will be accommodated at Passfield Hall in single bed-sitting rooms. There is hot and cold running water in each room.

Cost. The fee for tuition and residence, inclusive of gratuities, is £7 7s. 0d.

PROGRAMME

Tuesday, 17 April, 8-8.30 p.m. Inaugural Address. Speaker: Dr. John Burton. 8.30-9.30 p.m. *Fundamentals in Health Education.* Lecturer: Dr. W. Emrys Davies.

Wednesday, 18 April, 9.15-10.30 a.m. *Learning by Participation.* Lecturer: Dr. W. Emrys Davies. 11 a.m.-12.15 p.m. *Discussing Health Problems: The Case-Study Approach.* Lecturer: Dr. John Burton. 3.15-4 p.m. and 5-7 p.m. *The Selection and Use of Materials in Health Education.* Lecturer: Dr. W. Emrys Davies.

Thursday, 19 April, 9.30 a.m.-12 noon. *Mass Media in Health Education.* (i) *Material for Illiterate People.* (ii) *Films and their Use.* Lecturer: W. Sellers, O.B.E. 5-7 p.m. (iii) *Posters and Displays.* Lecturer: Dr. W. Emrys Davies. 8-9.30 p.m. Films.

Friday, 20 April, 9.15-10.30 a.m. *Public Health Problem and Health Education.* Lecturer: Dr. A. J. Dalzell-Ward. 11 a.m.-12.15 p.m. and 3.15-4.15 p.m. Group Discussions. 5-7 p.m. Preparation of Reports. 8-9 p.m. Plenary Session.

Applications should be directed to the Medical Director, Central Council for Health Education, Tavistock House, Tavistock Square, London, W.C. 1.

PASSING EVENTS : IN DIE VERBYGAAN

In the article Extracorporeal Hemodialysis, by Mario Battazzati, M.D. and Carlo Taddei, M.D., published in the *Journal* of 4 February, the bibliography now to hand should be as follows:

Alwall, N. *et al.* Acta med. scand. (1947): **128,** 317 and Suppl., **196,** 250. (1948): **131,** 237. (1949): **132,** 392, 572, 477 and Suppl., **229,** 1. (1950): **137,** 233 and **138,** 246. Acta chir. scand. (1954): **108,** 95.

Battezzati, M., Taddei, C. *et al.* Minerva med. (1953): **102,** 2001, 2007 and 2010. (1954): **100,** 1615, 1619, 1629, 1634 and 1638.

Dr. Roderick Maggs, M.B., Ch.B., M.R.C.P.E., F.R.F.P.S., D.C.H. has changed his address and now conducts his consulting paediatric practice from Room 510 Southern Life Building, St. George's Street, Cape Town. Telephone: rooms 2-7247, residence 69-2580.

Dr. Roderick Maggs, M.B., Ch.B., M.R.C.P.E., F.R.F.P.S., D.C.H., het van adres verander, en praktiseer nou as konsulterende kinderaarts te Southern Lifegebou (Kamer 510), St. Georgesstraat, Kaapstad. Telefoon: kamers 2-7247, woning 69-2580.

BOOK REVIEWS : BOEKRESENSIES

NEUROGLIA

Neuroglia Morphology and Function. By Paul Glees, M.A., D.Phil., M.D. Pp. 111 + xii, with 44 illustrations. 25s. Oxford: Blackwell Scientific Publications. 1955.

Contents: 1. Introduction. 2. Historical Introduction. 3. Comparative Studies of Neuroglia. 4. Macroglia. 5. Microglia. 6. Neuroglia in Tissue Cultures. 7. General Conclusions.

This book is a good one. Happily, it is a short one to boot, and the author's claims for it are correspondingly meagre. His 'fairly extensive review of the literature' makes reference to every international figure in this field—just the kind of 'broad-spectrum' bibliography that is hard to find in medical text-books now. In relating past work to his own extensive investigations and other very up-to-date researches, he produces not only a beautiful thumbnail sketch of his specialty but clears up the confusion of names which the earlier workers left behind them. A second claim for the book, expressed modestly as the hope that its morphological studies will be useful to biochemists and others exploring cerebral function, is guaranteed by the exhaustive and impartially presented text. It is regrettable that the specialized nature of the subject will limit the book's readers, and also a pity that the author had not space to discuss commonplace oddities encountered by the general pathologist, such as corpora amylaceae or myelin figures.

The book has minor faults: typographical errors (explanations on page 92 might need a tissue culturist to rewrite), incomplete and incorrect keys to illustrations, and a curious transcription from Brain and Greenfield (1950) on page 61 which if true is 'curiouser'.

J.A.H.C.

A TEXT-BOOK OF MEDICINE

A Text-book of Medicine. Edited by Russell L. Cecil, M.D., Sc.D. and Robert F. Loeb, M.D., Sc.D., D. Hon.Causa., LL.D. Ninth Edition. Pp. 1786, with illustrations. Philadelphia and London: W. B. Saunders Company. 1955.

Contents: 1. The Infectious Diseases. 2. Diseases of Allergy. 3. Collagen Diseases. 4. Diseases due to Physical Agents. 5. Diseases due to Chemical Agents. 6. Deficiency Diseases. 7. Diseases of Metabolism. 8. Diseases of the Ductless Glands. 9. Diseases of the Digestive System. 10. Diseases of the Respiratory System. 11. Diseases of the Kidneys. 12. Diseases of the Spleen and Reticulo-endothelial System. 13. Diseases of the Blood. 14. Diseases of the Cardiovascular System. 15. Diseases of the Locomotor System. 16. Diseases of the Nervous System. Appendix. Index.

It is not easy to review a standard text-book. For this reason it has almost become the practice among reviewers to weigh it, or measure it, evaluate the cost per page, or perform some other arithmetical feat upon it. Regarding 'Cecil', now in its 9th edition, it is certainly too heavy to hold in the hand, but the most interesting arithmetic seems to me to be the number of contributors, which is over 200. One lone representative from Britain appears (Dr. Desmond Curran). In a book of this type the contributions are necessarily uneven. It would be easy to criticise the sections on sarcoidosis, on dyschondroplasia (this term should not be used), on renal calculi and so on. Thus no mention is made of the importance of bed rest, paraplegia and hypercalcuria in the development of renal stones, while the treatment is inadequately considered. The syndrome of prediabetes is not mentioned. Nevertheless this book in many ways is extremely good—the introduction (by Fuller Albright) to the section on Endocrinology bodes fair to becoming a classic; the chapter on adrenal diseases, gout and the deficiency diseases are particularly good. This edition actually includes mention of meticorten in the chapter on rheumatoid arthritis.

The paper is excellent and the type also good, which makes for pleasant reading. The index is adequate, no misprints were found and there are some helpful figures. There are a few colour-photos —Dr. Spies seems to have done well here (in the section on nutrition); he even has a boy with rickets in colour! The smallest colour-picture ever must be that of Lactrodactus mactans (black widow spider); incidentally the button spider is missing.

Altogether this book must be considered one of the best general text-books of medicine. It is of particular use to students, and for the most part the articles can be recommended as being highly authoritative.

P.J.

THE SCIENTIFIC BASIS OF MEDICINE

British Postgraduate Medical Federation, University of London. Lectures on the Scientific Basis of Medicine. Volume III, 1953-1954. Pp. 366 + ix, with 7 illustrations. 35s. London: University of London, The Athlone Press. 1955.

Contents: 1. Science and History. 2. Biological Synthesis. 3. The Genetics of Some Biochemical Abnormalities. 4. Tissue Repair. 5. The Supporting System and its Disorders. 6. Hemispherectomy and the Localization of Function. 7. Anticholinesterases. 8. Acetylcholine and the Maintenance of the Cardiac Rhythm. 9. The Growth Hormone of the Anterior Pituitary Gland. 10. Stress and Thyroid Activity. 11. The Physiological Actions of the Sex Hormones. 12. Acid and Alkaline Phosphatase in Disease. 13. Body Water Control. 14. Reactions to Bacterial Invasion. 15. Antiviral Immunity. 16. The Action of Bacterial Enzymes on Immunizing Antigens. 17. Causes of Failure in Antibiotic Therapy. 18. Antimalarial Drugs. 19. Chemotherapy of Cancer. 20. The Scientific Approach to Dermatology. 21. Experimental Psychopathology.

The spectacular advances in medicine today come from research in the basic sciences, and even the most hardened clinician must be

ready to prate knowingly of lysozymes and globulins, and to face hydroxyl groups without flinching. Unfortunately the field is now so wide that no one can have first-hand knowledge of more than a corner or two and, to many, its whole expanse is unknown. It is in showing what exciting work is going on that the lectures on the Scientific Basis of Medicine, organized by the Postgraduate Medical Federation, are so valuable. Short of attending them personally one cannot do better than to read this book and its two predecessors. Each lecture is short enough to be read at the kind of sitting that is all one gets in these days, and most of the subjects are so absorbing that the book has the quality of a volume of detective stories. It is excellently adapted to reading in bed.

The matter of these lectures is difficult to criticize because of its diversity; this reviewer, at any rate, could give an expert opinion on about two pages of the 400. He is thus well qualified to review the book for others as ill-endowed, if such exist. The first essential for an expert expounding his own subject to non-specialists is that he should be intelligible, and this almost all the lecturers are. Some, like Paton on the anti-cholinesterases, are unexpectedly entertaining as well. Even those with the most specialized interests should find something of value in this book, and general medical readers bent on improving their minds as well as those preparing for higher examinations should put it on their lists.

P.B.

BOOKS RECEIVED : BOEKE ONTVANG

BOOKS RECEIVED RECENTLY IN THE WITWATERSRAND MEDICAL LIBRARY

Bergin, K. G. Aviation medicine. Bristol. Wright. 1949.
Bernal, J. D. The physical basis of life. London. Routledge. 1951.
Birch, C. A. The house physician's handbook. Edinburgh. Livingstone. 1955.
Bryan, J. E. Public relations in medical practice. Baltimore. Williams & Wilkins. 1954.
Chandler, A. C. Introduction to parasitology. 9 ed. New York. Wiley. 1955.
Coon, C. S. The history of man. London. Cape. 1955.
Cruickshank, W. M., ed. Cerebral palsy. Syracuse U.P. 1955.
Greene, E. C. Anatomy of the rat. New York. Hafner. 1935 reprinted 1955. (American Philosophical Society. Trans. n.s. 27 1935.)
Harris, G. W. Neural control of the pituitary gland. London. Arnold. 1955.
Hinsie, L. E. Psychiatric dictionary. 2 ed. Oxford U.P. 1953.
Johannesburg Conference on Social Work. Report of the Johannesburg Conference on Social Work, 9-14 October 1950. Johannesburg. Social Affairs Department. (1955.)
Kotinsky, R. ed. Community programs for mental health. Cambridge. Mass. Published for the Commonwealth Fund by Harvard U.P. 1955.
Lawrence, J. H. Polycythemia. New York. Grune & Stratton. 1955.
Parsons, L. The influence of Harvey and his contemporaries on paediatrics. London. Headley. 1950.
Satake, Y. Secretion of adrenaline and sympathins. Tokyo. Nanzando. 1955.
Scientific Council for Africa South of the Sahara. List of scientific societies in Africa South of the Sahara. London. Published under the sponsorship of the Commission for Technical Cooperation in Africa South of the Sahara. (1955.)
Sodeman, W. A., ed. Pathologic physiology. Philadelphia. Saunders. 1950 repr. 1952.
Thomson, St. C. Diseases of the nose and throat. 6 ed. London. Cassell. 1955.
Williams, R. H. ed. Textbook of endocrinology. 2 ed. Philadelphia. Saunders. 1955.

Journals Received for the First Time in 1955

Acta Tuberculosea Scandinavica.
Arquivos Del Instituto Cardiologia De Mexico.
Australasian Annals of Medicine.
British Abstracts of Medical Sciences.
Central African Journal of Medicine.
Danish Medical Bulletin.
Disease-a-Month.
Journal of Chronic Diseases.
Journal of Biophysical and Biochemical Cytology.

CORRESPONDENCE : BRIEWERUBRIEK

HOW TO IMPROVE THE JOURNAL

To the Editor: Within recent months we all have received copies of new South African medical publications and journals, appealing for subscribers, and all endeavouring to aid in the postgraduate education of the general practitioner, but so far I have not seen one of them containing the essential information which will help to keep him informed on the latest developments in medicine. With the rapid progress of modern medicine there is a dire need for such a medical journal, which will give the busy general practitioner a concise conception of the latest developments, drugs, treatments, etc.

The problem of the conscientious general practitioner, with his much discussed dwindling status, is where to find the time even to peruse a small portion of the medical writings. As a family doctor, he must attend patients at his rooms, visit them at home, know their most intimate details in order to act as a front-line psychiatrist, make night calls, and attend maternity cases; and (though in a graceful manner he has been excluded by the authorities from most hospitals, to the detriment of the public as a whole, and to the profession in particular) he must still visit patients in nursing homes.

As a member of human society he must try and devote a portion of his time to his neglected family, attend social and civic functions, and, in order to avoid being dubbed mentally deficient, read daily newspapers and lay publications, so that he can carry on an intelligent conversation with patients and friends on the latest atomic bombs, murders, jewel robberies, and political developments in his own and foreign countries. In order to keep physically fit, he must still find time to relax, sleep, eat, and take part in sporting activities. In addition to all these activities he must keep up his postgraduate studies by attending medical meetings, revision courses and medical congresses and, most important of all, by regularly reading at least one or two medical journals. The most important of these will be the *South African Medical Journal*.

It is time, therefore, that we took stock of our *Journal* to see, whether it can be of more value to the hard-pressed G.P. This is not a criticism of the *Journal*, and I wish to congratulate you on the high standard of the *Journal*, and on the excellent job of work you are doing, but I wish to make the following suggestions, as a general practitioner, who has only time to read the *Journal* regularly, and one or two other medical publications:

Every week the *Journal* should contain one revision article, with latest theories and treatments.

Each original article should have an intelligent summary (not the present concise summary), with discussions and conclusions.

Each issue of the *Journal* should contain a section dealing with the latest new drugs and appliances, available in South Africa and overseas; news of medical society activities and answers to queries of South African practitioners.

But most important, once every 4 weeks, an enlarged edition should be published, devoted almost entirely to abstracts of

medical literature. This could perhaps be done by publication, by arrangement, of the abstracts, contained in the *Journal of the American Medical Association*, queries and answers of interest, as contained in the *British Medical Journal* and the *Journal of the American Medical Association*, summaries of important original articles contained in various British and American medical journals and publications, and latest drugs, antibiotics, treatments and appliances available in America and Europe.

In this way the family doctor will be kept abreast of the latest developments, without depending for a great deal of his knowledge on the brochures and blotting-paper advertisements that are liberally supplied by the various drug houses. The extra cost of such an informative edition could be met by extra advertisements and by increasing the annual subscription, to which few subscribers would object. There may be technical, and copyright difficulties in publishing articles from foreign journals, but with the universal goodwill in international medicine, these can surely be overcome.

H. J. Sutherland

'Sherwood'
Woodgate Road
Plumstead
31 January 1956

EXPERIENCE WITH MITRAL VALVOTOMY AT GROOTE SCHUUR HOSPITAL

To the Editor: I have read again the detailed article[1] under this title, published in your esteemed *Journal* on 26 November 1955. The heading of the article implies personal experience with the 75 cases reviewed and therefore criticism cannot be directed at the facts but only at some of the erroneous conclusions. From the surgical point of view much of the experience and advice detailed there is out of date and not in keeping with current thoracic surgical practice.

The authors state: 'The adequacy of the valvotomy has a bearing on the result. The result is adequate if the orifice is widened to over 3 sq. cm. *Safe partial valvotomy* has been the method of *choice*'. The authors' definition of a wide opening is not shared by Sir Russell Brock,[2] who states that the size after valvotomy is fair when over 3 sq. cm., good when over 4 sq. cm., and excellent when there is a full opening admitting 2 fingers. Again Goodwin *et al.*,[3] from the Postgraduate School at Hammersmith, state the opening is only fair if 3½ sq. cm. in area and good if greater than 4 sq. cm. Charles P. Bailey,[4] from Philadelphia, states the opening should be at least 2 fingers.

Mr. Phillips' digital expertness as exemplified by the statement that 'very few valves require cutting, as a wide valvotomy is usually achieved by finger pressure alone' is unique. As long ago as 1952 Brock[5] stated that in 28% of his cases it was necessary to use a knife, and on a recent visit to Guy's Hospital I found the knife was used more frequently.

Jordaan[6] writing in this *Journal* in 1954 stated that he found it necessary to use a Brock's knife in 38% of cases. Bailey,[7] on an experience of 811 cases states in 1955 that in only 37·5% of cases was he able to obtain an adequate split digitally, and the balance—i.e. 62·5%—needed instrumental help. Robert P. Glover,[8] also in 1955, stated at the International Meeting of Cardio-Vascular Surgeons in Detroit that he was able to obtain an adequate split only in 25% of cases; in 50% the finger was combined with the knife and in 25% of cases it was essential to use the knife. In the light of these figures Mr. Phillips' use of the knife in only 3 out of the last 48 cases—i.e. 6%—is surprising.

To quote again: 'In the ideal case valvotomy takes only a few seconds with minimal disturbance'. This happy state of affairs is indeed common when the finger only is used and an inadequate partial split along the commissures obtained. Mr. Phillips makes no mention of sub-valvular stenosis in his series. I presume, from our own experience, that this is because the cross union of the chordae *below* the cusp edge can only be felt and separated when a wide opening to the mitral ring is obtained. In our first 80 cases we were not aware of this added obstruction, but in the past 18 months, where necessary, we have made strenuous efforts to separate these adherent chordae as suggested in the past 2 years by most authors; e.g. Holmes Sellors[9] states that 'after valvotomy the finger is passed down within the fan of the chordae to free any cross union'. Bailey[4] says, 'Sub-valvular finger dissection of the chordae is essential'. Personally we still find this procedure fraught with difficulty and the fear of producing regurgitation.

The authors are uncertain 'whether re-stenosis will recur if the valve is not completely opened'. It should be noted that only 10 of their cases have been followed for 2 years. I look forward to publication of their results in the surviving cases after a further 5 years; I believe from our experience and from the literature that unless the valve commissures are split or cut widely the risk of recurrence is great. As Paul Wood[10] has pointed out, stenosis only occurs many years after infection. Recurrence after inadequate surgery will take place in a fair percentage of cases after 3-15 years.

I fully agree that not only is mitral regurgitation more likely to result from instrumental valvotomy but also that regurgitation surgically induced is dangerous. I have lost 2 patients with this post-operative condition.

This criticism of inadequate splitting, I should like to add, is chronological. At first it was felt that a circumference of 7·5 cm. and an area of 3 sq. cm. would be adequate. A follow-up of these cases, however, showed deterioration in so many cases that wide commissurotomy to the mitral ring is now attempted as a routine, even if not always obtained because of technical difficulties.

As an illustration of the changed attitude towards the need for instrumental valvotomy it may be recalled that in 1953 I reported, with Mr. D. Fuller,[11] our first 50 cases subjected to mitral valvotomy. In only one was the knife used effectively; like the authors, we were also satisfied with an area of 3 sq. cm. and splitting of only the antero-lateral commissure. The altered surgical approach is reflected in the following table abstrated from our case records:

	Finger only	*Finger and knife*	*Knife only*	*Failures*
50 cases, 1952–1953	41	0	1	7
50 cases in 1955	20	15	14	3

The 3 recent 'failures' would have been classified as 'fair' in 1953.

I would also like to substantiate the authors' conclusions that mitral valvotomy in suitable subjects adequately performed does give dramatic relief from the most distressing and incapacitating symptoms.

David Adler

Florence Nightingale Building
22 Kotze Street
Johannesburg

23 January 1956

1. Schrire, V., Vogelpoel, L., Phillips, W. and Nellen, M. (1955): S. Afr. Med. J., **29**, 1108 (26 November).
2. Baker, C. and Brock, R. C. (1955): Brit. Med. J., **2**, 986.
3. Goodwin, J. F. *et al.* (1955): *Ibid.*, **2**, 573.
4. Bailey, C. P. (1955): *Surgery of the Heart*, p. 585. London: Henry Kimpton.
5. Baker, C. and Brock, R. C. (1952): Brit. Med. J., **1**, 1043.
6. Jordaan, M. (1954): S. Afr. Med. J., **28**, 391.
7. Bailey, C. P. (1955): *Op. cit.*[4] p. 579.
8. Glover, R. P. (1955): *Cardiovascular Surgery*, p. 197. Philadelphia: Saunders.
9. Holmes Sellors, T. (1955): Brit. Med. Bull., **2**, 210.
10. Wood, P. (1954): Brit. Med. J., **1**, 1121.
11. Adler, D. and Fuller, D. (1953): S. Afr. Med. J., **27**, 1176.

RELAXANTS IN ANAESTHESIA

To the Editor: Your editorial[1] of 19 November 1955 points out that the mortality of anaesthesia in a representative cross-section of the United States increased 6-fold with the use of the muscle relaxants in the period 1948-52 (Beecher and Todd[2]). No inference was made by you as to the relative position in South Africa, but it is common knowledge throughout the medical profession that a similar state of affairs exists here. The fact that nurses do not administer anaesthetics in South Africa does not imply that our mortality figures may be less alarming, since the data collated by Beecher and Todd do not show that the death rate of the nurse anaesthetists was any worse than that of the specialists.

In your issue of 17 December 1955 Dr. Roberts[3] takes you to task. Your editorial seems to him unfair, in that 'it presents only one side of the picture, presenting a few data culled at random

from Beecher and Todd's paper, and failing to mention any of the very cogent criticisms of their findings that have appeared in the medical press in the past 16 months'. I do not agree with this. The data were sufficiently startling to warrant your action in bringing them to the notice of the profession. If anyone is unfair, it seems to me to be your correspondent, who quotes you out of context and somewhat inaccurately.

Beecher and Todd state that they do not *know*[4] (their italics, overlooked by your correspondent!) that 'curare' has killed a single patient. This is a statement on their part, not an admission, as quoted by Dr. Roberts. Of course they do not *know*. No anaesthetist, surgeon or doctor, or indeed any professional person, can afford to confess the unvarnished truth. In the matter of anaesthetic data, the confessor of a fatal error lays himself open to a charge of culpable homicide. But the inference cannot be escaped that there is every probability that the relaxants have been accompanied by a shocking rise in mortality—in minor as well as major surgery.

It will be admitted that the majority of anaesthetics in this country are not given by specialists. Keeping in mind the number administered by general practitioners in town and country, and by trainees and interns in hospitals, only the minority are given by specialists. Yet your correspondent states that 'in practice no patient should die from hypoxia due to the use of relaxants or respiratory depressant drugs'. Unfortunately they do die, and I think you may be pardoned for taking the liberty of warning those who administer anaesthetics of this inescapable fact.

It is obvious in the light of the data collated by Beecher and Todd that the muscle relaxants *per se* had to be incriminated. These two investigators could come to no other logical conclusion. I am of strong opinion that sudden cardiac arrest can be produced by a massive dose of a long-acting relaxant, and it is a fact that Flaxedil, even in the usual doses, causes tachycardia and increased heart-output. Nevertheless, I certainly agree with your experienced correspondent that the cause of death, when normal doses of relaxants are used, is more often than not due to the grave effects of hypoventilation—hypoxia and hypercapnia.

There is no evidence to date of inherent toxicity in Scoline, apart from its specific action in relaxing voluntary muscle. It is my firm contention, therefore, that if the long-acting relaxants are abandoned by the inexperienced, the incidence and dangers of prolonged hypoventilation will be greatly diminished, and mortality will thereby be curbed.

H. H. Samson

602 Ingram's Corner
Twist Street
Johannesburg
27 January 1956

1. Editorial (1955): S. Afr. Med. J., **29**, 1084.
2. Beecher, H. K. and Todd, D. P. (1954): Ann. Surg., **140**, 2.
3. Roberts, F. W. (1955): S. Afr. Med. J., **29**, 1211.
4. Beecher, H. K. and Todd, D. P., *op. cit.*, p. 14.

WHITHER MEDICINE

To the Editor: Dr. Alan Sichel is to be congratulated on his courageous and timely message to members of the Medical Association, although it has produced a spate of undesired publicity in both the lay press and the *South African Medical Journal*, and at least one personal and deplorable letter of criticism to the *Journal*.

It has been explained to me that 'Times are changing' and that is the reason for the financial 'new look' in our profession. Times have been changing since the world began, not only in the lives of its peoples, and particularly in technical medicine, but surely not in the humanities.

Having devoted considerable thought over at least the past 20 years to this 'new look', I have come to the conclusion that the tragedy of medical practice today is that the patient is fast becoming a ledger entry rather than a person in trouble. There is no profession or craft that does not provide the meed of reward for work conscientiously and well done, both spiritually and materially. Where material reward for work done looms too large, the quality of work, usually done in a hurry and possibly without thought, must deteriorate. This is axiomatic of all work, not only medical practice. Those of the profession who are dissatisfied with the remuneration for work done are not compelled to practice medicine; other fields of activity, with greater reward, would appear to be more fitting to their outlook.

Few people are today satisfied with the economics of the walk of life they have chosen. This is, however, no reason why we should whittle away the time-honoured dignity of our profession for a mess of pottage.

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27 January 1956

RADIOTHERAPY IN CANCER OF THE BLADDER

To the Editor: In your issue of 7 January Mr. de Klerk[1] condemns the figures presented by Dr. Ralston Paterson[2] concerning carcinoma of the bladder as useless, and in support of his contention implies that the Institute at Manchester has relied only on cystoscopic and histological diagnosis. He further makes the quite astounding statement that in Great Britain adequate transurethral biopsy and bimanual examination under anaesthetic is 'unfortunately' not yet widely practised.

Both these methods of examination are widely practised in Great Britain and, what is more, have been so practised for certainly as long, and as widely, as in any other country. Full cooperation in all institutions in Great Britain exists between urologists and radiotherapists in the assessment of bladder cancer, and all available methods of investigation are widely used. This includes chemical and bacteriological examination of the urine, cystoscopy, pyelogram, cystogram, bimanual examination under anaesthetic, transurethral biopsy and full blood chemistry.

The masterly work of Masina and Dukes[3] in placing the classification of epithelial tumours of the bladder on a sound footing demands this. Not only do urologists and radiotherapists assess the depth to which the growth has spread through the bladder wall—and this can in no pre-operative method be 100% accurate—but they also need to know the site and size of the lesion, the condition of the surrounding mucous membrane, and the histology of the lesion, apart from the making of a full clinical examination of perivesical tissues, as well as skiagrams of chest and pelvis for any possible metastases, before a final decision is made on what method of treatment will benefit the patient most. In fact in the Ogies-Ward method of extra-vesical implantation of radon seeds for bladder cancer, in a suitably adjusted lesion, opening of the peritoneum is practised to ascertain if any peritoneal spread has occurred.

Jewitt and Lewis[4] reported in 1949 on cases of total cystectomy, graded according to the extent of the infiltration of bladder muscle, and showed that if the tumour had spread up to halfway through the bladder musculature the prognosis was good, 90% of cases being still alive; if the tumour had spread more than halfway through the bladder musculature 90% of cases had died; this in post-operative specimens. How very much more difficult must the decision be in pre-operative assessment when partial cystectomy is contemplated!

Partial cystectomy, except for the small early lesion ideally situated, e.g. the fundus, is very nearly an obsolete operation. By its very nature it negates all the principles of cancer surgery.

Whether or no Mr. de Klerk, is convinced by Dr. Ralston Paterson's figures, radiotherapy including deep and super-voltage X-rays, radium and the many radio-active isotopes (bromine, sodium, cobalt, tantalum), each having its own specific sphere of usefulness, is already playing the major part in the attack on bladder cancer in Great Britain, whose urologists and radiotherapists working as a team can compare in experience and excellence with any in the world and whose results in bladder cancer stand comparison with any.

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1. de Klerk, J. N. (1956): S. Afr. Med. J., **30**, 24.
2. Paterson, R. (1955): *Ibid.*, **29**, 1215 (24 December).
3. Masina and Dukes: (1949): 5th Annual Meeting of British Association of Urological Surgeons, 30 June 1949. Brit. J. Urol., **21**, 275.
4. E. W. Riches: Reporting on results of Jewett and Lewis in discussion at above meeting.